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A MANUAL OF

THE

PRACTICE OF MEDICINE

BY

GEORGE ROE LOCKWOOD, M. D.

Professor of Practice in the Woman's Medical College of the New York Infirmary; Attending Physician to the Colored Hospital and to the City (late Charity) Hospital; Pathologist to the French Hospital; Member of the New York Academy of Medicine, of the Pathological Society, of the Clinical Society, etc.

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TO

HORACE DENNETT

AS A TOKEN OF RESPECT AND AFFECTION

THIS BOOK IS RESPECTFULLY DEDICATED

BY

THE AUTHOR

PREFACE.

It has been the aim of the author to present in this manual the essential facts and principles of the practice of medicine in a concise and available form. It is hoped that the work will meet the requirements of those who heretofore have been obliged to resort to the larger works of reference with which medical literature is so well supplied.

In the arrangement of the subject-matter the admirable classification of Osler has been adopted with but a few unimportant modifications.

Acknowledgment of the author's indebtedness is hereby made to those writers from whose articles illustrations have been taken for use in this manual, credit in each case being given in the text. The author also desires to extend his thanks to Mr. Thomas F. Dagney, of Mr. Saunders' publication rooms, for the preparation of the index and for valuable suggestions of a varied character while the manual was going through the press.

NOVEMBER, 1895.

CONTENTS.

I. THE INFECTIOUS DISEASES.

PAGE
Typhoid Fever 17
Typhus Fever 46
Relapsing Fever 51
Small-pox 55
Vaccinia 64
Varicella 65
Scarlet Fever 66
Measles 76
Rubella 81
Epidemic Parotitis 83
Whooping-cough 85
Epidemic Influenza 89
Dengue 94
Epidemic Cerebro-spinal Meningitis 95
Diphtheria 102
Erysipelas 117
Pyæmia 121
Septicæmia 124
Cholera 126
Yellow Fever 133
Syphilis 137
    Acquired Syphilis 138
    Hereditary Syphilis 145
Acute Miliary Tuberculosis 148
Malarial Fever 152
Anthrax 163
Hydrophobia 165
Tetanus 169
Leprosy 173
Glanders 176
Actinomycosis 179
Milk Sickness 181
Weil's Disease 181

II. DISEASES OF THE CIRCULATORY SYSTEM.

1. Diseases of the Pericardium 183
    Pericarditis 183
    Purulent Pericarditis 192

PAGE
Chronic Adhesive Pericarditis 193
Tubercular Pericarditis 195
Cancerous Pericarditis 196
Hydropericardium 196
Hæmopericardium 197
Pneumopericardium 197
2. Diseases of the Heart 198
Hypertrophy and Dilatation 198
Hypertrophy 202
Dilatation 206
Acute Endocarditis 212
Malignant Endocarditis 219
Chronic Endocarditis 223
Atheroma 225
Mitral Incompetency 226
Mitral Stenosis 230
Aortic Regurgitation 234
Aortic Stenosis 239
Tricuspid Regurgitation 241
Tricuspid Stenosis 242
Pulmonary Regurgitation 242
Pulmonary Stenosis 243
3. Diseases of the Myocardium 249
Acute Myocarditis 249
Chronic Myocarditis 251
Syphilitic Myocarditis 252
Degeneration of the Myocardium 253
Aneurysm of the Heart 259
Rupture of the Heart 260
4. Neuroses of the Heart 261
Palpitation 261
Tremor Cordis 263
Intermittent Action 263
Tachycardia 263
Brachycardia 264
Angina Pectoris 266
Pseudo-angina 269
Exophthalmic Goitre 271
5. Congenital Malformations 275
6. Diseases of the Arteries 277
Arterio-sclerosis 277
Syphilitic Arteritis 281
Aneurysm 281
Aneurysm of the Abdominal Aorta 291

III. DISEASES OF THE RESPIRATORY SYSTEM.

1. Diseases of the Larynx 292
Spasm of the Larynx 292
Acute Catarrhal Laryngitis 294
Chronic Catarrhal Laryngitis 296

PAGE
- Membranous Laryngitis 297
- Tubercular Laryngitis 298
- Syphilitic Laryngitis 300
- Œdematous Laryngitis 301

2. Diseases of the Bronchi 302
   - Acute Catarrhal Bronchitis 302
   - Acute Croupous Bronchitis 306
   - Chronic Catarrhal Bronchitis 308
   - Chronic Croupous Bronchitis 310
   - Bronchiectasis 310
   - Asthma 313
3. Diseases of the Lungs 318
   - Circulatory Disturbances 318
     - Congestion of the Lungs 318
     - Œdema of the Lungs 319
     - Pulmonary Hemorrhage 320
       - Broncho-pulmonary Hemorrhage, or Hæmoptysis 320
       - Pulmonory Apoplexy 323
   - Lobar Pneumonia 324
   - Broncho-pneumonia 341
     - Broncho-pneumonia in Adults 348
   - Interstitial Pneumonia 352
   - Interlobular Emphysema 355
   - Vesicular Emphysema 356
   - Gangrene of the Lung 362
   - Abscess of the Lung 364
   - Syphilis of the Lung 365
   - New Growths of the Lung 366
   - Echinococcus Cysts of the Lung 368
   - Tubercular Inflammations in General 369
   - Tubercular Diseases of the Lung 373
     - Acute Pulmonary Tuberculosis 373
     - Chronic Pulmonary Tuberculosis 377
     - Acute Pulmonary Phthisis 382
     - Chronic Pulmonary Phthisis 386
4. Diseases of the Pleura 400
   - Fibrinous or Dry Pleurisy 400
   - Pleurisy with Effusion 403
   - Purulent Pleurisy 411
   - Chronic Pleurisy 416
   - Tubercular Pleurisy 418
   - Pneumothorax 420
   - New Growths of the Pleura 424
   - Hydrothorax 425
   - Hæmothorax 426
5. Diseases of the Mediastinum 427
   - Lymphadenitis 427
   - Mediastinal Tumors 428
   - Abscess of the Mediastinum 430
   - Emphysema of the Mediastinum 430
   - Mediastinal Hæmatoma 431
   - Diseases of the Thymus Gland 431

IV. DISEASES OF THE DIGESTIVE SYSTEM.

PAGE
1. Diseases of the Œsophagus 432
    Acute Œsophagitis 432
    Chronic Œsophagitis 433
    Stenosis of the Œsophagus 433
        Cicatricial Stenosis 434
        Cancerous Stricture 435
        Spasmodic Stenosis 437
    Dilatations and Diverticula 438
    Paralysis of the Œsophagus 439
    Rupture of the Œsophagus 439
    Varix of the Œsophagus 439
2. Diseases of the Stomach 440
    Acute Catarrhal Gastritis 440
    Toxic Gastritis 442
    Acute Croupous Gastritis 443
    Acute Suppurative Gastritis 444
    Mycotic and Parasitic Gastritis 444
    Chronic Catarrhal Gastritis 445
    Dilatation of the Stomach 452
    Ulcer of the Stomach 456
    Cancer of the Stomach 464
    Non-cancerous Tumors of the Stomach 471
    Hemorrhage from the Stomach 472
    Neuroses of the Stomach 475
        Nervous Dyspepsia 475
        Hypersecretion and Hyperacidity 476
        Gastralgia 477
        Nervous Vomiting 479
        Peristaltic Unrest 479
        Rumination 480
3. Diseases of the Intestines 480
    Morning Diarrhœa 480
    Acute Catarrhal Enteritis 481
    Chronic Catarrhal Enteritis 483
    Phlegmonous Enteritis 486
    Pseudo-membranous Enteritis 486
    Mucous Colitis 487
    Diarrhœal Diseases of Children 489
        Acute Dyspeptic Diarrhœa 490
        Acute Entero-colitis 490
        Cholera Infantum 492
    Cholera Morbus 496
    Dysentery 497
        Acute Catarrhal Dysentery 498
        Tropical or Amœbic Dysentery 499
        Acute Croupous Dysentery 501
        Chronic Dysentery 504
    Appendicitis 505
        Acute Catarrhal Appendicitis 506
        Acute Suppurative Appendicitis 508

PAGE
Gangrenous Appendicitis 513
Chronic Appendicitis 515
Ulceration of the Intestine 516
Cancer of the Intestine 521
Cancer of the Rectum 523
Cancer of the Caput Coli 524
Cancer of the Duodenum 525
Non-cancerous Tumors of the Intestine 525
Intestinal Obstruction 525
Hemorrhage from the Intestine 531
Fecal Accumulation 533
Amyloid Degeneration of the Intestine 536
4. Diseases of the Peritoneum 536
Acute Peritonitis 536
Acute Peritoneal Sepsis 537
Acute Diffuse Peritonitis 538
Acute Circumscribed Peritonitis 542
Chronic Peritonitis 545
Chronic Hemorrhagic Peritonitis 547
Tubercular Inflammations of the Peritoneum 547
Acute Tuberculosis of the Peritoneum 547
Acute Tubercular Peritonitis 548
Chronic Tubercular Peritonitis 549
Cancer of the Peritoneum 551
Ascites 553
5. Diseases of the Liver 556
Functional Disturbances of the Liver 556
Circulatory Disturbances of the Liver 559
Diseases of the Capsule of the Liver 560
Acute Perihepatitis 560
Chronic Fibrinous Perihepatitis 561
Syphilitic Perihepatitis 562
Acute Parenchymatous Hepatitis 562
Cirrhosis of the Liver 565
Atrophic Cirrhosis 565
Hypertrophic Cirrhosis 570
Syphilitic Cirrhosis 571
Abscess of the Liver 573
Tubercular Disease of the Liver 579
New Growths of the Liver 579
Hydatid of the Liver 583
Fatty Liver 586
Amyloid Liver 588
Jaundice 589
Acute Febrile Jaundice 592
Catarrhal Jaundice 592
Cholelithiasis 594
Cancer of the Gall-ducts 602
Diseases of the Blood-vessels of the Liver 603
6. Diseases of the Pancreas 605
Hemorrhage 605
Acute Hemorrhagic Pancreatitis 606

PAGE
Gangrenous Pancreatitis 607
Suppurative Pancreatitis 608
Chronic Pancreatitis 608
Pancreatic Cysts 609
Cancer of the Pancreas 610

V. DISEASES OF THE KIDNEY.

Congenital Malformations of the Kidneys 612
Movable Kidney 612
Anomalies of the Urinary Secretion 614
    Albuminuria 614
    Hæmaturia 617
    Hæmoglobinuria 619
    Pyuria 621
    Peptonuria 621
    Phosphaturia 622
    Lithæmia; Uricæmia 622
    Oxaluria 624
    Chyluria 624
    Indicanuria 624
    Glycosuria 625
    Lipuria 625
    Acetonuria 625
Acute Congestion of the Kidneys 625
Chronic Congestion of the Kidneys 626
Acute Degeneration of the Kidneys 627
Chronic Degeneration of the Kidneys 628
Acute Exudative Nephritis 629
Acute Diffuse Nephritis 632
Chronic Bright's Disease 635
    Chronic Diffuse Nephritis with Exudation 635
    Chronic Diffuse Nephritis without Exudation 639
Waxy Degeneration of the Kidney 647
Tubercular Diseases of the Kidney 648
    Tuberculosis of the Kidney 648
    Tubercular Pyelonephritis 648
Suppurative Disease of the Kidney 650
Tumors of the Kidney 653
Cysts of the Kidney 655
Pyelitis 656
Hydronephrosis 657
Nephrolithiasis 659
Perinephritic Abscess 666

VI. CONSTITUTIONAL DISEASES.

Acute Articular Rheumatism 667
Pseudo-Rheumatism 675
Chronic Articular Rheumatism 675
Muscular Rheumatism 677
Gout 678

PAGE
Arthritis Deformans 686
Diabetes Mellitus 689
Glycosuria 696
Diabetes Insipidus 697
Scurvy 698
Rickets 701
Purpuric Diseases 704
  Symptomatic Purpura 705
  Purpura Rheumatica 705
  Purpura Hæmorrhagica 706
Hæmophilia 709

VII. DISEASES OF THE BLOOD AND THE LYMPHATIC GLANDS.

Anæmia 712
  Secondary Anæmia 712
  Chlorosis 716
  Pernicious Anæmia 718
Leucocytosis 722
Leukæmia 722
Pseudo-leukæmia 727
Addison's Disease 731
Tuberculosis of the Lymph-glands 733

VIII. DISEASES OF THE NERVOUS SYSTEM.

1. Diseases of the Membranes of the Brain 736
(*a*) Diseases of the Dura Mater 736
  Acute External Pachymeningitis 736
  Acute Internal Pachymeningitis 736
  Chronic Internal Pachymeningitis 737
  Syphilitic Pachymeningitis 738
(*b*) Diseases of the Pia Mater 739
  Tubercular Meningitis 739
  Acute Non-tubercular Meningitis 741
  Syphilitic Meningitis 744
  Chronic Meningitis 744
  Meningeal Hemorrhage 745
2. Diseases of the Blood-vessels of the Brain 747
  Congestion 747
  Anæmia 748
  Œdema 749
  Cerebral Hemorrhage 749
  Embolism of the Cerebral Arteries 755
  Thrombosis of the Cerebral Arteries 758
  Aneurysm of the Cerebral Arteries 758
  Thrombosis of the Venous Sinuses 760
  Cerebral Endarteritis 761
3. Diseases of the Brain-substance 764

PAGE
Cerebral Localization 764
Abscess of the Brain 769
Tumors of the Brain 772
Cerebral Atrophy in Children 778
Cerebro-spinal Sclerosis 781
General Paresis 782
Chronic Hydrocephalus 784
Syphilis of the Brain 785
4. Diseases of the Spinal Cord 788
(*a*) Affections of the Meninges 788
Diseases of the Dura Mater 788
Diseases of the Pia Mater 790
Acute Leptomeningitis 790
Chronic Leptomeningitis 792
Hemorrhage into the Spinal Membranes 793
(*b*) Diseases of the Blood-vessels 794
Anæmia 794
Hyperæmia 794
Hemorrhage into the Cord 795
Caisson Disease 796
(*c*) Diseases of the Substance of the Cord 797
Acute Anterior Poliomyelitis 797
Anterior Poliomyelitis in Children 798
Anterior Poliomyelitis in Adults 802
Subacute and Chronic Poliomyelitis 803
Progressive Muscular Atrophy 804
Amyotrophic Lateral Sclerosis 807
Bulbar Paralysis 808
Ophthalmoplegia 810
Lateral Sclerosis 810
Locomotor Ataxia 813
Hereditary Ataxia 821
Ataxic Paraplegia 821
Myelitis, Acute and Chronic 822
Acute Ascending Paralysis 827
Syringo-myelia 828
Compression-myelitis 829
Tumors of the Spinal Cord 830
Brown-Séquard's Paralysis 830
5. Diseases of the Cranial Nerves 831
Olfactory Nerve 831
Optic Nerve 831
Third Nerve 832
Fourth Nerve 833
Fifth Nerve 833
Sixth Nerve 834
Seventh Nerve 835
Auditory Nerve 838
Glosso-pharyngeal Nerve 840
Pneumogastric Nerve 840
Spinal Accessory Nerve 844
Hypoglossal Nerve 846

PAGE
6. Diseases of the Peripheral Nerves 847
  Neuritis 847
    Localized Neuritis 847
    Multiple Neuritis 850
  Neuromata 855
  Neuralgia 856
7. General Nervous Diseases 860
  Infantile Convulsions 860
  Epilepsy 862
  Paralysis Agitans 869
  Acute Delirium 871
  Chorea 873
    Choreiform Affections 879
  Tetany 880
  Migraine 882
  Occupation-neuroses 884
  Neurasthenia 885
  Hysteria 887
  Sun-stroke 895
  Delirium Tremens 898
8. Vaso-motor and Trophic Disorders 899
  Raynaud's Disease 899
  Acute Circumscribed Œdema 900
  Facial Hemiatrophy 901
  Myxœdema and Cretinism 902
  Scleroderma 904
  Acromegaly 904

IX. DISEASES OF THE MUSCLES.

Myositis 906
Progressive Muscular Dystrophy 906
Paramyoclonus Multiplex 908
Thomsen's Disease 909

X. ANIMAL PARASITES.

Trematodes 910
Nematodes 911
  Ascaris Lumbricoides 911
  Oxyuris Vermicularis 911
  Anchylostoma Duodenale 912
  Trichocephalus Dispar 912
  Trichina Spiralis 913
  Filaria Sanguinis Hominis 914
Cestodes 915
Echinococcus Disease 917

A MANUAL

OF THE

PRACTICE OF MEDICINE.

I. THE INFECTIOUS DISEASES.

TYPHOID FEVER.

Definition and Synonyms.—Typhoid fever is an acute infectious disease caused by a specific bacillus, and is characterized anatomically by lesions of the intestinal and mesenteric glands and by enlargement of the spleen. The disease runs a febrile course of three or four weeks, with a characteristic eruption and systemic symptoms. *Synonyms:* Typhus fever (*German*); Abdominal typhus; Ileo-typhus; Enteric fever; Autumnal fever.

Etiology.—Typhoid fever is one of the most widely spread of all the infectious fevers; it occurs in all countries and in all climates, though it is more frequent in the temperate zones. It may occur at any time of the year, but it is most commonly seen in late summer and in early fall, hence the name "autumnal fever" which has sometimes been applied to it. It seems to occur with especial frequency after hot, dry summers.

The disease affects the sexes equally, although in hospital practice more cases are met with in men, because they more readily apply for hospital treatment.

The disease may occur at any age, but young adults between the ages of fifteen and twenty-five are especially susceptible. After the age of thirty-five the disease appears progressively infrequent.

There is in this disease, as in all infectious diseases, a great difference in personal susceptibility, some individuals being more readily infected than others who have been subjected to the same degree of exposure. In cities strangers are more susceptible to this fever than old inhabitants. The development of the disease after exposure is favored by any inflammatory condition of the intestine, the entrance of the germ into the lymphatics being favored by the epithelial desquamation resulting from the catarrhal process. A low and sickly condition of the general health does not seem to increase the susceptibility to infection. The disease appears in both epidemic and endemic forms. The epidemics are usually local, affecting a group of houses, an institution, or a part of a town. The longer the epidemic, the more difficult it often is to trace the source of infection. It appears as an endemic disease where it had previously existed as an epidemic, and it is endemic in almost all large cities. The source of infection is with difficulty traced in endemic cases.

The actual exciting cause of typhoid fever is now proved to be the infection of the patient by a specific germ known as the *bacillus typhosus*, or Eberth's bacillus. This germ is a short, mobile bacillus whose length is equal to one-third the diameter of a red blood-cell, and having rounded bulbous ends which often present a shining appearance, due rather to alterations in its protoplasm than to spore-growth, as was at first supposed. In its appearance and growth this germ closely resembles the *bacterium coli commune*, or ordinary colon-bacillus, from which it is hard to differentiate it. The typhoid bacillus grows with ease in almost every kind of nutritive media, and it possesses extraordinary vitality. It may persist in drinking-water or in the soil for weeks or for months, and may even increase in number. It grows with great rapidity in milk without altering its appearance or taste; and so great is its tenacity of life that it may remain imprisoned in ice for months without losing its virulent properties. In the accumulations of privy-vaults and sewers it finds conditions most favorable for its growth and activity. Cultures are killed by a temperature of 60° C., by carbolic acid (1 : 200), and by corrosive-sublimate solution (1 : 2500). Cultures resist drying for several days, but the growth of the bacilli is retarded by exposure to sunlight.

The bacillus obtains entrance to the body through the alimentary canal, and enters the intestinal lymphoid tissue probably through abrasions of the epithelial coat. It has been found in the lymphoid tissue of the intestines, in the mesenteric glands, the spleen, the liver, at times in the blood taken from the rose spots, and occasionally in the urine. It has been found also in some of the complicating lesions of the disease. The bacilli are found in clusters in the intestinal contents and the stools of patients, and are thrown out from the body in this way. They are not eliminated from the lungs or the skin.

Methods of Infection.—The disease is in no sense personally contagious, cases of typhoid being received into the general wards of hospitals without risk. The bacilli being cast off only in the dejecta of the patient, it is from the stools that danger of infection arises. If the stools are thoroughly disinfected and the bacilli are killed, there is no further risk of a spread of the infection. If the stools are not disinfected, however, the bacilli will live and thrive in them, and this infected sewage, draining into water-supplies, will spread the disease among those who drink of such water.

There are three ways by which the infection of typhoid may occur:

The first method is by direct infection from stools. While not common, infection has occurred among attendants on the sick and among those who have washed the soiled linen of typhoid patients, the germs being transferred from infected hands to the food, and thus obtaining entrance to the body.

The second method of infection is by contamination of the water-supply. This is the usual source of infection, and it explains the origin of epidemics of the disease that occur from time to time in towns, in institutions, and in villages. Contamination of drinking-water with filth and sewage will not produce the disease unless to such sewage is added the specific germ. Interesting investigations of epidemics frequently show their origin in the contamination of the water-supply by the dejecta of a single typhoid patient, even though months may have elapsed between the infection of the sewage and the consequent contamination of the water-supply. The source of contamination is most easily traced in small epidemics, and examples of epidemics in hotels, villages, and towns so traced are to be found reported in full in medical literature. In the same way the infection may be conveyed by impure ice, after the thawing of which the germs regain their vitality.

The third method of infection is by means of milk, in which the bacilli readily thrive, and to which they are added by impure water used either to wash the cans or to dilute the milk.

There are reports of epidemics apparently caused by eating meat of diseased cattle, but this mode of infection is not yet definitely determined. Poor drainage, sewer-gas, and imperfect hygiene will not of themselves cause the disease: they only offer favorable conditions for the growth and development of the bacillus.

Pathology.—The lesions are divided into those essential to, and those complicating, the disease.

Essential Lesions.—The essential lesions consist in—1. Changes in the lymph-glands of the intestine; 2. Changes in the mesenteric glands; 3. Enlargement of the spleen.

1. *The changes in the intestinal lymphoid tissue* are seen in both the solitary and the agminated glands, but especially in the latter. They are most constant in Peyer's glands of the lower portion of the ileum, and they may appear here alone. In about one-third of the cases the glands of the cæcum and colon are affected.

Congestion and Hyperplasia.—The first change consists in the congestion and swelling of the lymph-follicles, noticed on the second day of the disease. Toward the end of the first week there is added an increase in number of the cellular elements, some cells resembling the ordinary lymphoid cells, while others are large and round with several nuclei. This hyperplasia further increases the size of the gland. The cellular increase is not entirely confined to the gland, but infiltrates the mucous membrane in its vicinity, small isolated foci being also seen in the muscular, the subserous, and even the serous coats of the intestine. In these masses of lymphoid cells the bacilli are constantly found. This cellular increase persists during the second week of the disease. In very mild cases, the lesion goes no further than this, but resolution occurs, the congestion disappears, the cells undergo fatty degeneration and absorption, and the gland assumes again its normal appearance.

Necrosis.—In most cases, however, the lesion progresses to such a degree that resolution becomes impossible. The swelling of the gland presses on its blood-vessels, and, cutting off its blood-supply, induces a condition of anæmia-necrosis, and in consequence of this condition and of the direct action of the bacilli upon the tissues the cells die and are cast off, either gradually, by a process of ulceration, or *en masse*, by a process of gangrene. In either case an ulcer is left, the walls and floor of which are composed of infiltrated glandular tissue, and by the further disintegration of these infiltrating cells the ulcer may increase in size and in depth. Such ulceration may perforate through the entire intestinal wall, or the separation of the slough may be the cause of hemorrhage. The process of ulceration occupies the third week of the disease.

Cicatrization.—During the fourth week the ulcer begins to cicatrize and the normal glandular elements are re-formed. In some cases an ulcer may cicatrize in some portions and extend in others. Cicatrization should be complete toward the close of the fourth week, but the ulcer may remain sluggish and inactive until the sixth or the eighth week.

2. *Changes in the mesenteric glands* are of the same nature and intensity as those in the intestine, the only difference being that the products of necrosis cannot be thrown off, but form foci of softened purulent matter containing bacilli. Small foci may eventually be absorbed, while larger ones may become dry and cheesy and enclosed by a fibrous capsule. At any time a fresh focus may rupture into the peritoneal cavity.

3. *The spleen* regularly becomes increased in size and harder in consistency. These changes proceed until the third week, after which the spleen becomes soft and pulpy and returns to its normal size. In rare cases the spleen becomes soft, but does not increase in size. Rupture or a gangrenous abscess of the spleen may occur.

COMPLICATING LESIONS.—1. *Peritonitis.*—This condition may be caused in a variety of ways—either from perforation of the intestine by an ulcer, from rupture of a softened mesenteric gland, or from rupture, abscess, or infarction of the spleen. In some cases no cause can be found for the peritonitis.

2. *Catarrhal or Croupous Enteritis.*—Severe inflamma-

tions are rare, though a mild catarrhal enteritis is commonly seen.

3. *Parotitis.*—This condition may occur during the second or third week, and may proceed to the formation of an abscess.

4. The *liver* becomes hyperæmic and increased in size. The cells become swollen and coarsely granular. There may be foci of lymphoid cells in the substance of the organ.

5. The *kidneys* show parenchymatous degeneration of the cells of the convoluted tubules. In rare cases there may be acute nephritis. There may be small foci of infiltrating lymphoid cells which may proceed to suppuration, causing so-called "miliary abscesses." In these cellular foci Eberth's bacilli are found; they may be present also in the urine in these cases.

6. The *heart-muscle* is soft and flabby and may be the seat of waxy degeneration. There may be degeneration of any of the voluntary muscles. In rare cases pericarditis with effusion of serum or of pus is found.

7. The *pharynx* or the *larynx* is often the seat of a catarrhal or croupous inflammation. Œdema of the glottis may occur, and ulceration of the larynx occurs in a certain number of cases.

8. *Lungs.*—In nearly all cases there is some degree of hypostatic congestion. In other cases there is bronchitis with zones of peribronchial pneumonia. This is a serious lesion. Mild degrees of bronchitis are commonly seen. Gangrene or abscess of the lung may be found, and pleurisy with effusion occurs in a small number of the cases.

There may be thrombosis of some of the larger *veins*, especially of the femoral; less frequently there is thrombosis of the cerebral sinuses.

Symptoms.—As the symptoms are so complex and the clinical aspect of the disease so varied, it seems best to consider each symptom at first in detail, and then to give a general description of their clinical grouping.

Fever.—In a typical case the temperature should run a self-limited course of four weeks' duration, each week possessing characteristics of its own. During the first week the temperature gradually mounts, higher in the evening than in the morning, and higher each day than on the day previous. The maximum, which is reached by the end of the week, is between 103° and 104° F. The lowest morning temperature is between 6 and 8 A. M.; the highest evening temperature is between 6 and 9 P. M.

During the second week the temperature remains fairly stationary; there are morning remissions, but these are slight.

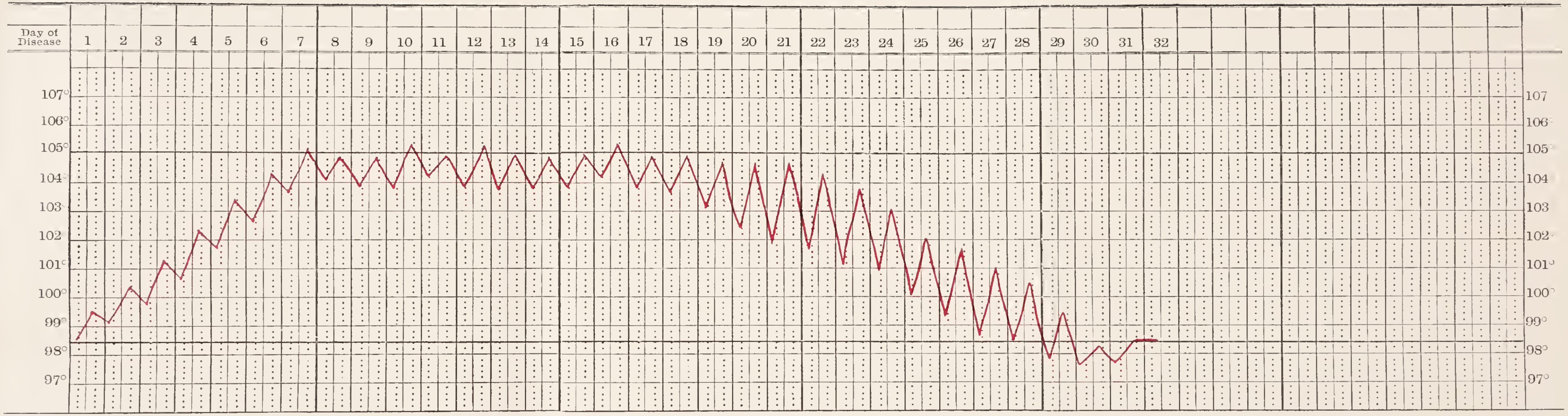
The temperature in the first part of the third week continues like that of the second week, but toward the close of the third week the morning remissions become more marked, the evening rise remaining the same.

During the fourth week the morning remissions become more marked, while the evening exacerbations decrease; toward the close of the week the morning temperature becomes normal, and, the evening rise becoming progressively less, the evening temperature becomes also normal and the case is completed.

This typical temperature is depicted on Plate I. While this temperature curve is typical of typhoid fever, such a regular schematic chart is seldom met with in actual practice, variations in the course being exceedingly common. The commonest variations of each week will be separately considered.

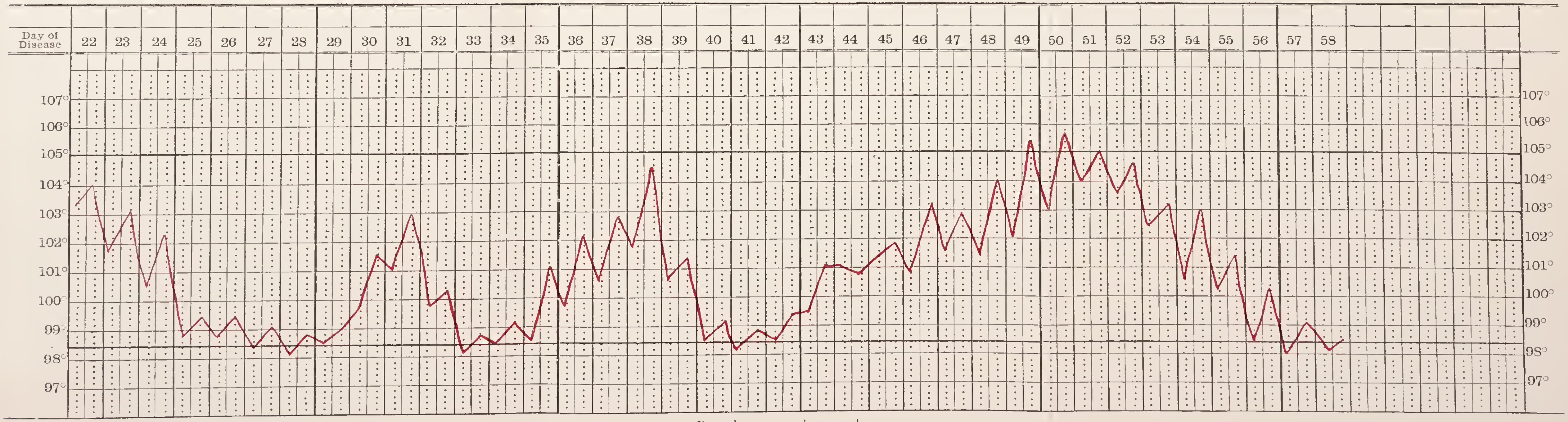
Variations in the First Week.—(*a*) It is seldom that we obtain the temperature of the first few days, as the disease begins so insidiously that it is not until the fourth or fifth day, as a rule, that the patient comes under observation.

TYPHOID FEVER. PLATE I.



Schematic temperature.

TYPHOID FEVER. PLATE 3.



Recrudescences and a true relapse.

(*b*) Some cases plunge at once into their fever, the onset being sudden, the temperature attaining its maximum upon the second or third day. This sudden onset is frequently accompanied by a chill. (*c*) In some cases the temperature of the first week is markedly remittent, the evening rise being frequently preceded by a chill and followed by sweating. Such cases are difficult to diagnose from malarial fever.

Exceptions to the Second and Third Weeks.—(*a*) The temperature may fall to normal by lysis at the close of the second week. These are the cases in which the lesion in the glands does not progress to ulceration. (*b*) During the second and third weeks the fever may be slight—between 99° and 101° F.—and the morning temperature may even be normal. This may indicate a mild attack if the other symptoms be mild in proportion, but if the other symptoms be severe the low temperature is not a good sign. These cases with slight fever seem especially liable to relapses. (*c*) During the third week the temperature may remain steadily high with but slight diurnal variations. In bad cases the temperature may remain steadily high, persisting during the fourth, fifth, sixth, seventh, and eighth weeks. Such cases are apt to be fatal. In some cases the temperature suddenly mounts up before death, reaching a height of 109° to 111° F. (Plate 2, Fig. 1).

Exceptions to the Fourth Week.—The temperature may fall somewhat toward the end of the fourth week, but may still continue a little elevated, running between 99°, 100°, and 101° F. until the sixth, seventh, eighth, or ninth week. With this slight, steady temperature there may be profuse sweating. These symptoms seem due to unhealed intestinal ulcers.

In some cases a slight evening rise of temperature to 99° or 100° F. may be noticed during the fifth and sixth weeks, without other unfavorable symptoms. This rise may be due either to a depressed nervous state or to some insidious complication. A sudden fall in the temperature with a subsequent rise is significant of an intestinal hemorrhage. The more severe the hemorrhage the more decided the initial fall of temperature (Plate 2, Fig. 2).

The further modifications in the temperature caused by complications, and the relapses, will be considered under separate headings.

Pulse.—During the first week the pulse is full, dicrotic, and about 100. During the second and third weeks it becomes quicker and feebler in direct proportion to the gravity of the case. A pulse persistently over 140 is of serious import. In some cases the pulse is feeble without being rapid. The weakness of the heart's action is of more consequence than mere rapidity, and may endanger the life of the patient, especially during the third and fourth weeks. In some cases there may be attacks of syncope or even collapse, in either of which the patient may die. In other cases the feebleness of the heart allows of venous congestion and cyanosis, which further endanger the life of the patient. From the poor circulation thrombi may occur in any of the larger veins. A sudden marked increase in the rapidity and feebleness of the pulse indicates, as a rule, perforation or hemorrhage.

Gastro-intestinal Symptoms.—The *appetite* is lost early in the disease, and does not return until convalescence.

The *tongue* at first is moist and coated. In severe cases it has a tendency to become dry, due rather to the severity of the disease than to the fever. In very severe cases the tongue becomes dry and brown, and brownish crusts, or sordes, collect upon the teeth and gums, interfering with articulation and deglutition. This is a bad sign.

The *pharynx* is usually inflamed during the first week of the disease.

Nausea and *vomiting* may occur at any time in the disease, but they are not usually severe if the diet and medication be judicious. In some cases, however, vomiting is so severe as to interfere with the feeding of the patient, and it may even cause death by exhaustion.

Diarrhœa is usually present, although in some places, as in New York, and in certain epidemics constipation is the rule. The diarrhœa depends less upon the extent of the ulcerations than upon the severity of the associated enteritis, and it is rarely marked until after the beginning of the second week. It may last a few days only or may continue throughout the disease, or it may alternate with constipation. The stools are abundant, thin, offensive, and of a grayish-yellow color, and are described as suggestive of pea soup. It is a true fecal diarrhœa, resembling the normal contents of the small intestine. The passages vary in number from two to five daily, more frequent passages being rare. The common occurrence of constipation as a symptom must always be borne in mind.

Tympanites develops in the second week in a great majority of cases, and it is due to paresis of the inflamed intestinal wall interfering with peristalsis. Tympanites usually causes bulging of the abdominal wall, although in some cases rigidity on palpation is alone detected. When once developed it is apt to persist throughout the disease. In moderate degree it does no more actual harm than to render the patient uncomfortable, but if excessive it displaces the diaphragm upward, interferes with the action of heart and lungs, excites nausea and vomiting, and increases the danger of perforation. Tympanites is a sign of serious import.

Pain and *tenderness* over the abdomen are commonly observed, and are usually more pronounced in the right iliac fossa. These symptoms are of no diagnostic significance.

Gurgling in the right iliac fossa is too common in other diseases to be distinctive.

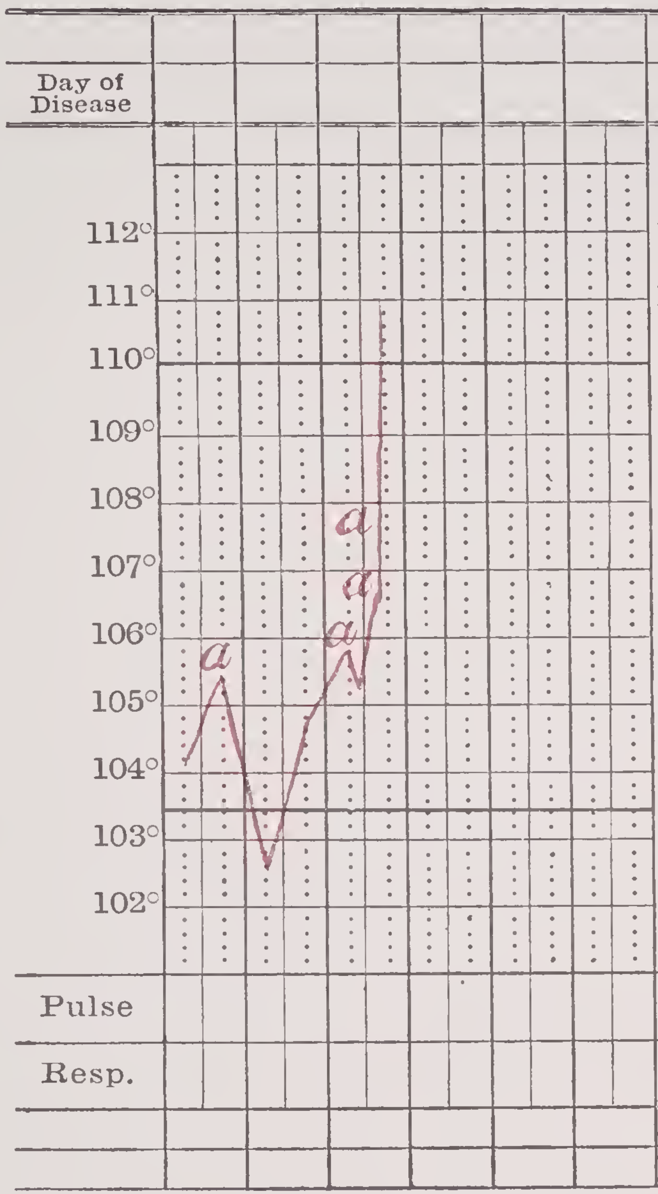
Hemorrhages from the bowels are a serious symptom. It is important to distinguish the sources of bleeding.

(1) There may appear slight hemorrhages during the first ten days of the disease, coming not from ulcers, for none exist at so early a date, but from the congested mucous membrane. They are trifling in amount, and indicate merely a severe degree of complicating enteritis.

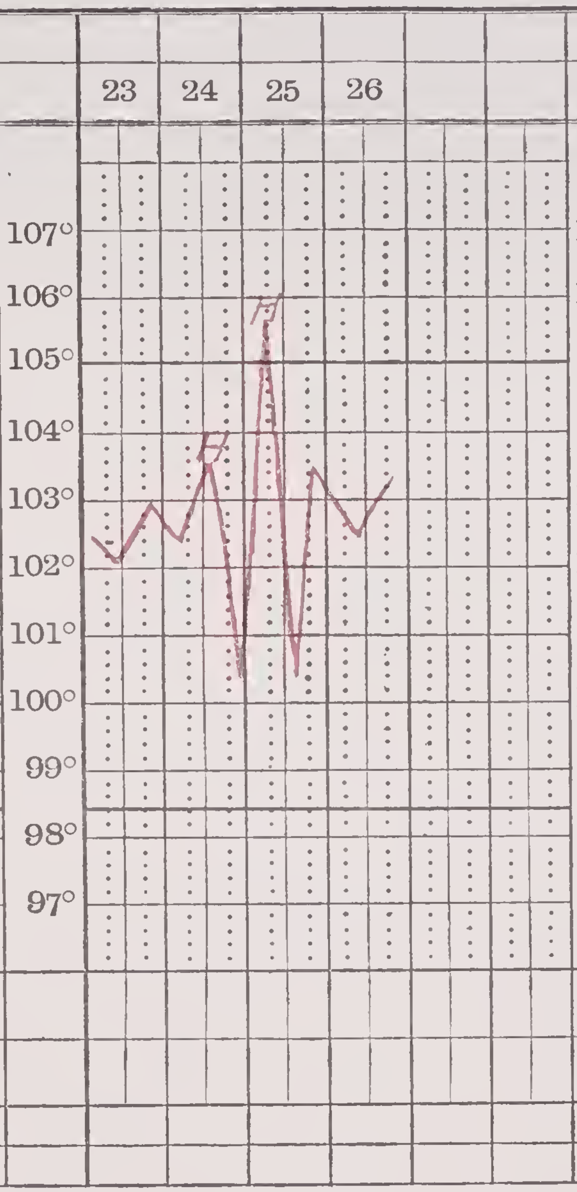
(2) The characteristic hemorrhages of the disease occur after the close of the second week, and are caused by the separation of the sloughs from the intestinal ulcerations. They occur in about 5 per cent. of cases, and are always of grave significance, for not only is the hemorrhage frequently fatal in itself, but it also denotes extensive and deep intestinal ulceration. The hemorrhage varies in amount from a few drops to several pints or more. When once a hemorrhage has occurred, others are liable to follow. The blood passed by the rectum may be fluid and red, or dark and clotted, or even tarry in appearance, according to its amount and the length of time it has been retained in the bowel. In some cases of rapidly fatal hemorrhage death may ensue before any blood at all has been passed. The symptoms caused by a severe hemorrhage appear suddenly—faintness, syncope, even collapse, a rapid and feeble pulse, great dyspnœa with restlessness (the so-called "air-hunger"), great pallor, and cold extremities. The mind, if clouded before, usually becomes clear and active. The temperature, as shown on Plate 2, Fig. 2, undergoes a fall of several degrees, perhaps to normal or even to subnormal, and this is followed usually by a subsequent rise to a higher plane than before the hemorrhage. From such a hemorrhage the patient may die in acute anæmia. In some rare cases the hemorrhage seems to exert a beneficial effect upon the

TYPHOID FEVER.

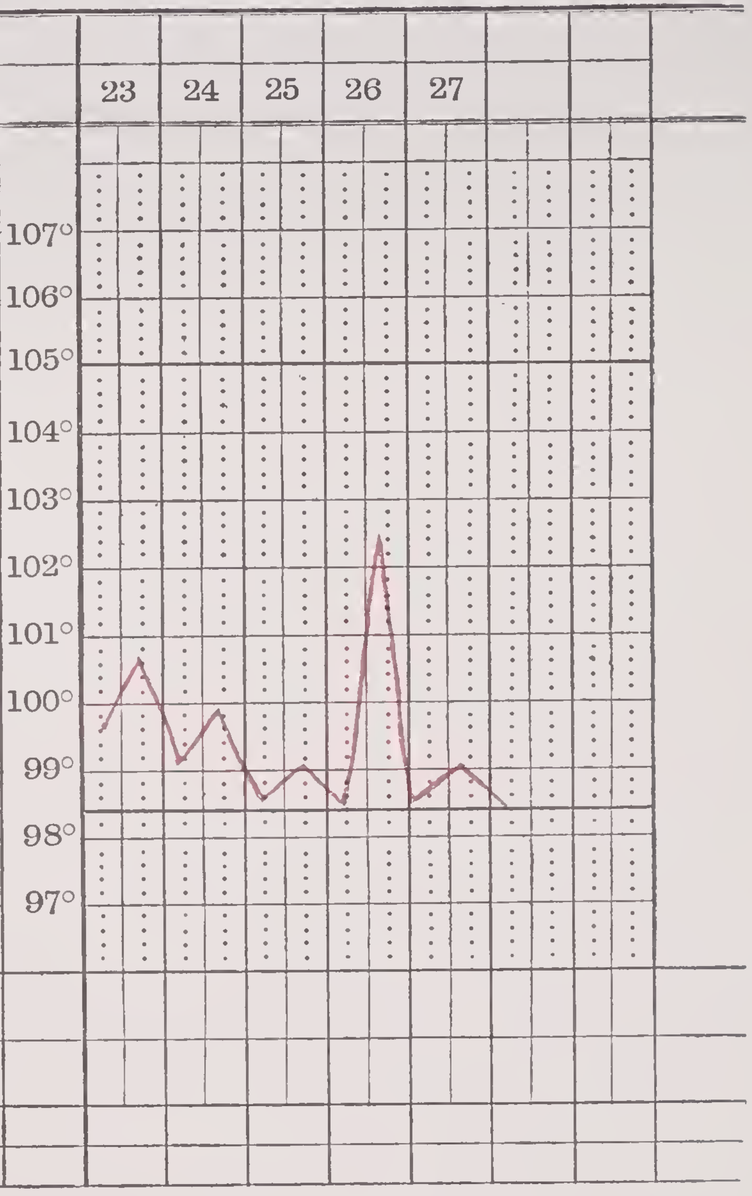
PLATE 2.



1. Ante-mortem hyperpyrexia
(*a* indicates administration of antipyrine).



2. Fall in temperature after
hemorrhage.



3. Short pseudo-relapse.

course of the disease. Subsequent hemorrhages may follow at intervals.

(3) During convalescence there may be small repeated hemorrhages without other unfavorable symptoms. These hemorrhages come from sluggish ulcers which have not yet cicatrized.

Nervous Symptoms.—During the first week headache is usually a pronounced symptom. It is usually dull in character, although in rare cases it may be severe, sharp, and associated with photophobia and convulsive twitchings, closely resembling in this respect the headache of meningitis. During the first week there are apt to be sleeplessness and restlessness at night. Associated with the headache there is often nose-bleed during the first week.

During the second week the headache and restlessness give way to a mental apathy which is exceedingly characteristic. The face is utterly without vivacity, with a vacant, listless expression. The pupils are generally dilated. The patients answer questions correctly, although with apparent lack of effort and interest, and often respond only to repeated and persistent questioning. If left alone, they lie quietly and do not seem to take any concern about themselves. This condition of apathy is in sharp contrast to the animated, cheerful condition of acute miliary tuberculosis, and is a strong point of differential diagnosis between the two diseases. It is important to remember that in rare cases this apathy is not seen.

Delirium is seen in the majority of cases, varying in degree from confusion of ideas to acute mania; it is, however, less commonly seen than formerly, owing to improved therapeutics. During the first week there may occur a delirium which is apt to be acute and violent. This condition, however, is not common. The commonest form of delirium, which appears after the second week of the disease, is of the low muttering variety, the patient lying quietly and talking incoherently. This form more usually appears only at night, although it may in severe cases last into the daytime as well. There may be a disposition of the patient to get out of bed, requiring careful watching. This delirium often alternates with periods of stupor which may become more profound, passing into semi-coma or even coma. In a few cases this form of delirium alternates with a more active and noisy variety. In many cases there appears no delirium at all, and the number of these cases seems to increase with the increasing applications of the hydropathic and antipyretic treatment of the disease.

In all cases muscular prostration is noticeable from the first and increases with the disease. There may be muscular weakness of the bladder, causing retention of urine. The passage of urine should always be inquired about, as the patient may not speak of it because of the mental apathy of the disease. There may be weakness of the sphincter ani with incontinence of feces.

In bad cases there are twitchings and automatic movements of the muscles, especially of the hands. These symptoms, frequently spoken of as "subsultus tendinum" and "carphologia," belong to the latter part of the disease. In rarer cases convulsive movements and muscular rigidity are seen in the first week of the disease, associated with the acute headache already alluded to.

Hyperæsthesia of the skin, which is occasionally met with, may be extreme.

Spleen.—The spleen is almost invariably increased in size, the enlargement being usually appreciable by the close of the first week. After the third week it diminishes in size. In rare cases the spleen may remain normal in size should there be depletion by severe diarrhœa or by hemorrhage. The enlargement of the spleen is more accurately determined by palpation than by percussion, as the splenic area is apt to be obscured by tympanites. In the majority of cases the spleen is tender on palpation.

Urine.—The urine shows during the first week the changes commonly seen in fever, being concentrated and depositing urates on cooling. During the course of the disease it is apt to contain a little albumin and casts from parenchymatous degeneration of the kidney, which degeneration, however, is never very severe. Ehrlich describes a color reaction which he considers characteristic of typhoid fever. To employ the test two solutions are prepared, one a ½ per cent. solution of sodium nitrite, the other a ½ per cent. solution of hydrochloric acid saturated with sulphanilic acid; 40 parts of the first solution are mixed with 1 part of the second, and equal parts of this mixture and urine are thoroughly shaken in a test-tube. Upon the addition of ammonia a brownish-red reaction is observed, whereas in normal urine a brownish-yellow, without any reddish, tinge is observed. This reaction is not obtained after the third week of the disease; but, while present during the earlier stages of the fever, it is not considered a pathognomonic test, as it may be seen in other diseases, such as acute tuberculosis or measles.

Eruption.—The eruption of typhoid fever is very characteristic. It consists of small round spots, about pin-head in size, of a rose-pink color which disappears on pressure, the spots being slightly elevated. In severe cases they may be hemorrhagic. They appear usually from the seventh to the twelfth day, although they may be seen as early as the third and as late as the twentieth day. They are present in typical relapses. They appear in successive crops, each crop lasting two or three days, but they do not appear after the third week. They usually are seen on the abdomen, but may be found on the chest, thighs, and back as well, and in rare cases may be more generally distributed. The eruption is usually scanty. There may be only two or three spots, and unless care is exercised they may be overlooked entirely.

Course of the Disease.—*Incubation.*—The period of incubation lasts a week or ten days, during which time there are apt to be indefinite symptoms—headache, lassitude, and slight digestive disturbances. These symptoms are neither characteristic nor well marked.

The *onset* is usually gradual, so that it is hard to tell exactly the first appearance of the disease; in practice, however, the first noted advent of fever is considered the definite time of onset.

During the *first week* the temperature rises from day to day; there are sore throat and probably epistaxis, dull headache with sleeplessness and restlessness, possibly a little wandering at night, and a slight apathetic condition during the day. The appetite is wholly lost. There may be nausea or vomiting; the bowels may be either loose or confined. The pulse varies from 80 to 100 and is of fair force. By the end of the week the spleen is felt enlarged and the eruption appears on the abdomen. Patients differ considerably as to the severity of these symptoms and as to their reaction to them, some patients being much prostrated, while others even well advanced in the disease are around and out. These "walking cases" are more usually seen in hospital practice, and they greatly increase the mortality rate. These walking cases may not come under observation until hemorrhage or perforation has occurred.

Occasionally the onset is more acute. (*a*) In some cases the disease begins with a chill and a rapid rise of temperature. (*b*) In other cases there appear acute nervous symptoms resembling those of meningitis—headache, photophobia, rigidity of the neck, and muscular twitching. (*c*)

In other cases the onset of the disease is accompanied by severe bronchitis, which renders the diagnosis between typhoid fever and acute tuberculosis one of great difficulty. (*d*) In other cases the disease may be ushered in by severe nausea and vomiting, while in rare cases the symptoms of acute nephritis, with smoky urine containing albumin and casts, are the first observed.

During the *second week* the symptoms become aggravated; the temperature remains steadily high, and the headache gives way to mental apathy. There may be at night sleeplessness and slight delirium, which in severe cases continues at intervals into the day. The pulse is a little more rapid and feeble. The eruption is more apparent. The lips and tongue are apt to be dry. Prostration becomes more and more marked. There may be diarrhœa or constipation. There is apt to be meteorism. At the close of the week the patient may die with severe nervous symptoms or from an early hemorrhage or from perforation, while very mild cases may defervesce. These mild cases defervescing at the close of the second week are frequently spoken of as "aborted typhoids," and constitute the set of cases in which the intestinal lesion does not progress to ulceration.

During the *third week* the temperature remains high, but the morning remissions become more marked. The pulse is more rapid and feeble, with a tendency to heart failure in severe cases. Cerebral symptoms are prominent, the apathy alternating with muttering delirium. Emaciation and prostration are extreme. The tongue becomes dryish. There may be retention of urine and incontinence of feces. During the third week there is danger of perforation, peritonitis, hemorrhage, and pulmonary complications.

During the *fourth week* the temperature finally reaches the normal, the pulse becomes stronger, cerebral symptoms disappear, the tongue becomes moist and clean, and the patient complains only of hunger and weakness. In some cases the fourth week may show no improvement over the third, but the symptoms will continue into the fifth, sixth, or seventh week without mitigation. At the end of this time the symptoms may improve and the patient recover, or they may become more pronounced and lead to a fatal termination. In other cases the temperature begins to fall in the fourth week, but does not quite come down to normal, the patient continuing with a slight irregular fever for from one to three weeks longer. In severe cases this fever is accompanied by a continuance of general symptoms to some degree, the protraction of convalescence being due to unhealed intestinal ulcers. In others, although the fever continues in this way, the general symptoms rapidly disappear, and the patient improves daily. These cases seem due to post-febrile anæmia or to nerve-exhaustion.

Insidious Cases.—Besides this regular form we find cases of an irregular type. These cases are usually seen in epidemics of some size, and they run an insidious course. The pulse is but slightly affected, and the temperature is but slightly increased, varying between 99° and 100° F. In rare cases there is no fever. Headache, restlessness, sleeplessness, and prostration are noticed. There may be diarrhœa or constipation, but the enlargement of the spleen and the eruption are the only marked characteristic symptoms. These cases may not be sick enough to be in bed, or even in the house; the course is about four weeks, and while the prognosis is generally good, such light cases are as liable as the severer forms to hemorrhage and perforation.

Typhoid in Children.—Typhoid fever in children differs from that in adults in that there is less danger of hemorrhage or perforation, the pulse is more apt to be rapid and feeble, the cerebral symptoms are more prominent, and the temperature reaches its maximum earlier in the disease, is more remittent (hence often called "infantile remittent fever"), and often falls by crisis. The eruption is frequently slight or absent.

Typhoid in the Aged.—Typhoid fever in the aged runs an insidious and frequently a fatal course. The temperature is not so high as in adults, but the cerebral symptoms and prostration are more marked. Hypostatic pneumonia, heart failure, and nephritis are frequent complications.

Complications.—*Perforation* of the intestine, which is a most serious complication, occurs in 6 per cent. of cases. It is rare before the third week, although it has been noted on the eighth day. It may occur in convalescence. It is more apt to occur in severe cases frequently associated with hemorrhage. It seems to be favored by meteorism, vomiting, and dietetic errors. The intestinal contents entering the peritoneal cavity, there results perforating peritonitis.

In very severe perforations the first symptom is pain in the abdomen, followed by a lowering of the temperature with a subsequent rise, collapse, and death in a few hours, the condition of peritoneal septicæmia being produced. In less severe cases there is time for the production of a purulent peritonitis, which runs a course with typical symptoms, or, should the patient already be severely ill, the abdominal symptoms will be less marked, there may only be an increase of temperature, a more rapid and feeble pulse, and a hastened fatal termination. Diagnosis in such cases is difficult, especially if there previously existed abdominal tenderness and tympanites. Peritonitis with the above symptoms is also seen as the result of any of the other causes mentioned under the head of *lesions*. Such a non-perforative peritonitis runs a longer course and may be recovered from.

The other complications of typhoid are various, and only a brief mention of the most important can be made.

The pharynx is often inflamed in the first week. Otitis media may occur at any time. Parotitis may occur in the third or the fourth week and may proceed to suppuration.

The larynx may be the seat of a catarrhal inflammation or of ulceration. There may be œdema of the glottis or perichondritis of the arytenoid cartilages.

Bronchitis is common at any time of the disease.

Broncho-pneumonia and hypostatic pneumonia are seen in severe cases. They add but few subjective symptoms, being marked by the regular symptoms of the disease and by the apathetic condition of the patient. The temperature is apt to be raised, the pulse becomes weaker and quicker, and the physical signs are present.

Lobar pneumonia, gangrene of the lung, and pleurisy with effusion are occasionally seen.

Thrombosis of the femoral vein is frequently seen, accompanied by pain and œdema and by the cord-like feeling of the vein.

Catarrhal or croupous enteritis or colitis may occur in severe degree.

Albumin and casts are usually present in the urine in small amounts, from a mild form of acute degeneration of the kidney. Less frequently there is an acute exudative nephritis with diminution in the amount of urine and considerable amount of albumin and casts. In rare cases the urine may be suppressed. When the kidney is involved in this way there are not apt to be uræmic symptoms, but the temperature and pulse are affected for the worse, and the patient is apt to do badly.

Subcutaneous or subperiosteal abscesses may develop, in the latter case frequently associated with caries of the bone. In these abscesses Eberth's bacilli are found.

Malarial infection may complicate typhoid fever at any time. During the course of the fever the added infection gives a remittent character to the temperature curve, while if the malarial infection show itself during convalescence, the temperature is more characteristic, being markedly remittent or even intermittent. Examination of the blood shows the presence of the malarial organism.

Relapses and Recrudescences.—Relapses are seen in from 3 to 18 per cent. of the cases, varying in the different epidemics. A relapse is a second attack of typhoid with a repetition of the symptoms of the first attack, and is produced by a reinfection of the intestine from sloughs derived from some part above. The intermission between the original attack and the beginning of the relapse may extend to twenty-five days, the usual period being from five to eight days. It is not necessary to have an intervening period without high temperature, as the relapse may occur in the fourth week, before the temperature comes down to normal. There may be only one relapse or there may be several, becoming progressively milder and occurring at longer intervals. The symptoms appear sooner and are of shorter duration than those of the primary attack. The temperature attains its maximum on the third to the fifth day; the eruption is scanty, and as a rule appears on the third, fourth, or fifth day. The other symptoms are much less severe than those of the primary attack. The duration of the relapse is usually from ten to fourteen days, although it may last as long as thirty-nine days.

The conditions predisposing to relapse are not known, although it seems as if constipation were a predisposing factor.

Relapses are to be distinguished from the so-called "recrudescences," or temporary rises of temperature, of convalescence. These rises usually occur from dietetic errors or from over-exertion. We have a rise of temperature occurring suddenly and remaining for from one to five days without the enlargement of the spleen or the eruption.

These recrudescences do not add any element of danger: they only retard convalescence by just so much. Plate 2, Fig. 1, shows the temperature of recrudescence alone, and Plate 3 shows the temperature of recrudescence and relapse.

Convalescence is always slow and tedious, usually requiring months before the patient is in robust health again. Convalescence may be interrupted in several ways. There may be an irregular fever which will last for several weeks. There may be perforation or hemorrhage or peritonitis during convalescence. The mind may remain feeble for days or weeks. Some patients can hardly be said to convalesce at all, but remain feeble, emaciated, and anæmic, and die exhausted, the autopsy usually revealing extensive cicatrices. There may be peripheral neuritis with paralysis of groups of muscles. Gastro-intestinal symptoms may persist—vomiting, diarrhœa, or dysentery. The hair usually falls out during convalescence, but grows again.

The **prognosis** varies in hospital and private practice and in various epidemics. The mortality in mild epidemics is from 5 to 15 per cent.; in hospital practice, from 15 to 25 per cent. In the German military hospitals, when the patients are young and vigorous and are treated early, the mortality is from 1 to 8 per cent. A guarded prognosis must always be given, as mild cases may turn out badly and serious cases may recover. The liability to perforation, peritonitis, or hemorrhage lends unknown factors to each case. As a rule, patients with high and continuous temperature, or with an early involvement of nervous centres, as shown by muttering delirium with muscular tremors or with excessive meteorism and diarrhœa, are apt to do badly. The earlier a patient is treated and sent to bed, the better the prognosis. Fat, elderly people and those addicted to alcohol stand the disease badly. Perforation and peritonitis are nearly uniformly fatal. Recovery from a relapse is to be expected, as the symptoms are rarely severe, and perforation, hemorrhage, and peritonitis are infrequent.

Treatment.—*Prophylactic.*—Typhoid fever is largely a preventable disease, and the prophylactic treatment consists in destroying the germ where it is known to exist, and in preventing its admission to the human body. To accomplish the first object the following rules should be rigorously obeyed, and be persisted in until convalescence is thoroughly established:

The bed-linen and the clothes of the patient must be boiled for at least half an hour after being soaked in a strong antiseptic solution. The following are types of the disinfectant solution to be employed:

℞. Bichloride of mercury,	ʒij;
Potassium permanganate,	ʒij;
Water,	1 gallon.—M.

℞. Chloride of lime (best quality),	℥iv;
Water,	1 gallon.—M.

Those washing or handling soiled bed-linen must cleanse their hands frequently in one of these solutions, especially before eating.

The intestinal discharges and the vomited matters must be mixed thoroughly with sufficient disinfecting fluid for at least half an hour before being emptied from the vessel. From time to time disinfecting fluid must be poured down water-closets or privy-vaults. The discharges must not be emptied into any privy-vault that is near the water-supply.

The nates of the patient must be cleansed and disinfected thoroughly after each defecation.

To prevent the admission of the germ into the body the drainage, the sewage, and the water-supply must be sanitary. The source of every epidemic should be traced in the most painstaking manner, and means should be taken to avert future infection. During an epidemic drinking-water and milk should be boiled, and care should be exercised that ice taken from polluted water is not used. With perfect disinfection of the dejecta and with perfect drainage not allowing pollution of water-supplies, typhoid fever should never occur.

General Management.—The patient should be put to bed as early as possible, and be kept there until convalescence has been established for one week. The use of the bed-pan and urinal should be insisted on. The room should be large, airy, and moderately cool. Care should be taken to prevent bed-sores by keeping the sheets smooth and clean and by washing the patient's back morning and night with alcohol and tannic acid, and after careful drying dusting it with starch-powder or with bismuth. The mouth and the teeth should be kept scrupulously clean, to avoid infection of the middle ear and of the parotid gland. Moreover, by cleansing the mouth the liability to pneumonia and stomach fermentation is much lessened. For this purpose listerine and water (1 : 8) or boracic-acid solutions (1 : 24) are of service. The services of a good nurse should be obtained, as nursing is one of the most important factors in the treatment of the case. In severe cases two nurses are necessary.

The diet should be fluid and easily digestible, milk being the ideal food. If given properly, there are but very few patients who cannot take it. From three to four pints should be given in the twenty-four hours, in divided quantities at short intervals. As a practical rule, a glassful every two hours in the day and when awake at night will guide its administration in the majority of cases. Personal attention must be given to the feeding of the patient to know exactly how much is taken and how well it is borne. In case pure milk is not well borne, as shown by nausea or vomiting, or by the finding of undigested curds in the stools, the milk should be reduced in quantity or be diluted with lime-water or any of the aërated waters, or should be peptonized. In case these measures fail, the patient may be fed with koumiss or with matzoon. In the rare cases in which milk cannot be taken at all the reliable preparations of beef peptonoids, beef tea, white of egg with water, Nestlé's food, etc. may be given in its stead, although they are to be avoided if possible. Alcohol should not be given as a routine treatment, but only when especial indications for it arise.

Medical Treatment.—As there is no specific drug capable of exerting a direct cure for the disease, the treatment is mainly an expectant one, controlling those symptoms that exert a baneful effect on the patient, threaten his recovery, and cause him discomfort. The only internal treatment that seems to be of any service is the use of intestinal antiseptics for disinfecting the alimentary tract, to prevent fermentation and tympanites, and to guard, if possible, against auto-infection and relapses. The drugs used are salol, β-naphthol, bismuth salicylate, and creosote, and high enemata of weak tannic-acid solutions. These measures seem in some cases to be of service. Except for these, the treatment is entirely symptomatic.

Fever.—In the treatment of the fever there exists a diversity of opinion, some recommending its reduction as soon as a moderate point is reached (102.5° F.), others preferring the temperature to run its own course unless it reaches too high a degree (105° to 106° F.). It is certain that the mortality of typhoid has been lessened materially by antipyretic treatment, and that whereas a few patients seem to be no worse for their fever, in the great majority of cases the reduction of the fever is accompanied by an improvement of all the symptoms. It seems, then, that the best treatment is to keep the temperature reduced, provided this can be done without harm or risk to the patient. The best method of doing this is by the use of hydrotherapy, first insisted on by Brand, but modified to suit individual cases, no strict rule being rigorously applied.

Brand's original method consists in the immersion of the patient in a bath of 60° F. for fifteen minutes, repeated every three hours so long as the temperature is 102.2° F. by the rectum. During the bath cold water is applied to the head and the surface of the body is briskly rubbed. After the bath the patient is dried, wrapped in blankets, and given a hot alcoholic drink if chilliness is apparent. No internal antipyretics are employed.

Under the Brand treatment it is claimed that not only is the temperature reduced, but that there is also a tonic effect produced on the circulatory and nervous centres, the intellect becomes clearer, the stupor less marked, muscular twitchings disappear, and insomnia is lessened, the patient frequently falling into a refreshing sleep. Complications are rendered more infrequent, and, what is most important, the mortality is reduced to a minimum. Brand's latest statistics show but twelve deaths in 1223 cases, a mortality of but 1 per cent. Not a patient died who came under treatment prior to the fifth day. These statistics, however, are taken from German military hospitals, where the patients are young, robust, and are treated early. Ordinarily, under this treatment the mortality is about 7 per cent. No effect is claimed in reducing the duration of the disease nor in lessening the liabilty to relapses. The contraindications are intestinal hemorrhage, perforation or danger of perforation, and peritonitis. Bronchitis and pneumonia do not prevent the treatment.

While this extreme method may be applicable in military hospitals and in robust, insensitive patients, its rigorous employment in all cases has decided disadvantages and requires modification. As a general rule, then, the modified bath must be employed, the temperature being 80° to 90° F. at the commencement and being gradually reduced 10° F. by the addition of cold water. This bath should be given whenever the temperature is 102.5° F. or over, provided it be not more frequent than every three hours. Friction of the body and affusion of the head should be employed in all cases.

For nervous, sensitive patients who are in mortal dread of such a modified bath the wet pack may be employed. The bedding being protected by a rubber blanket, the patient is wrapped in wet sheets closely applied by brisk friction. From time to time the patient is sprinkled with cool water.

The most simple method of hydrotherapy is the sponging of the body with water or with water and alcohol. If done for ten or fifteen minutes this will cause a slight reduction in temperature, but the method is too inefficient to be of much benefit in severe cases.

The use of internal antipyretics is attended by many disadvantages, and is less frequently employed now than formerly. While the temperature may be reduced by these drugs, there is also a depression of the nervous and circulatory centres, so that stimulants may be required to overcome the effects of the drugs. The actual mortality seems to be slightly increased by their use. The drugs most frequently employed for this purpose are antipyrine (gr. x), phenacetine (gr. v), and antifebrine (gr. ij). They were formerly given in much larger doses than at present. Quinine is now given, not to reduce temperature, but for its tonic effect. Whatever antipyretic is used should be given in small doses repeated in two hours if necessary, and not too great a fall of temperature should be produced, the reduction of a degree or a degree and a half being usually sufficient.

The *pulse* should be watched carefully, with special regard to its weakness rather than its rapidity. Alcohol is the best stimulant, in the form of whiskey or champagne, and when indicated must be given freely until its effect is noticed, even if 8 to 12 ounces of whiskey be given in the twenty-four hours. Strychnine may be combined with the alcohol, and is of service. Digitalis may also be employed. In cases where there is a rapid feeble pulse with marked septic symptoms, large rectal enemata and of intestinal disinfectants often are of benefit.

Vomiting is best treated by regulating the diet. Bismuth and oxalate of cerium are occasionally serviceable. If vomiting seems to be due to the tympanites, the latter condition should be treated. In severe cases rectal feeding may have to be resorted to.

For the *diarrhœa* opium by the mouth or the rectum, with the addition of the ordinary astringent drugs, is to be given. The stools should be examined to see that the diarrhœa is not caused by undigested curds.

Constipation is to be treated in the first week by saline laxatives or by castor oil. Later in the disease enemata are preferable, so that the bowel is emptied every second day.

For the *tympanites* turpentine stupes or 5-minim doses of turpentine in capsule constitute the best treatment. Intestinal antiseptics, as salol, β-naphthol, or creosote, may be of service, while the insertion of a soft rectal tube may afford relief.

For *hemorrhages* the patient should be kept absolutely quiet, opium in full doses being given. The use of internal astringents does not seem to do good. Applications of ice to the abdomen may be employed. The diet should be restricted, although the patient may be given acid drinks and cracked ice. In case of severe hemorrhage external warmth and stimulants should be used. Subcutaneous injections of warm sterilized saline solutions may be given.

The treatment of *perforation* is that, in the first place, of collapse—by warmth to the body and free stimulation, while opium should be given in full doses. Should the perforation occur in a robust person or during convalescence, the question of laparotomy and closure of the perforation should be considered.

Peritonitis is to be treated by the cold abdominal coil and by opium in full doses. In selected cases laparotomy and drainage may be resorted to.

The *nervous symptoms* are best controlled by the hydropathic treatment, which, acting as a tonic on the nervous centres, reduces the restlessness, allays the delirium, and promotes sleep. Where this treatment can be employed drug sedatives are seldom needed. Where drugs are needed phenacetine (gr. v, q. 3 h.) will relieve the headache and restlessness, sulphonal (gr. x–xx) or chloralamide (gr. x–xv) will promote sleep. In severer cases opium may be necessary.

Complications are to be treated on general principles.

Treatment of convalescence is trying to physician and to patient alike. The greatest care should be exercised in the management of the diet, as the patients are ravenously hungry and clamor for food. The patient should be kept in bed for five days after the temperature is normal, and on a fluid diet—not necessarily, however, on milk. No solid food should be given for at least ten days. At the end of this time one solid meal may be given in the middle of the day, of chop or mutton with a baked potato, and afterward a gradual change to three meals a day may be allowed. There are cases where the evening temperature remains irregularly high. These cases are benefited by quinine and solid food. During convalescence attention should be given to the digestion and the bowels, and tonics should be administered. A change of air is to be recommended, and patients should not be allowed to return too soon to business and daily cares.

The *recrudescences* are to be treated by a milk diet and rest in bed while the fever remains high. The treatment of a relapse does not differ from that of the original attack.

TYPHUS FEVER.

Definition and Synonyms.—Typhus fever is an acute contagious disease with an acute onset, a characteristic eruption, and a febrile movement of about two weeks' duration. *Synonyms:* Cerebral typhus; Exanthematic typhus; Spotted, Camp, Jail, or Ship fever.

Etiology.—Typhus fever is endemic in England, Ireland, and Russia, and to a less extent in Poland, Galicia, and certain parts of Southeast Europe. A few cases occur every year in New York and Philadelphia. From time to time the disease occurs in other places in epidemics. These epidemics have usually followed wars or famines, the disease being regularly favored by the overcrowding of people in jails, houses, or camps, by poor hygiene, by starvation, and by filth. It never arises spontaneously, but always from some previous case. It is one of the most highly contagious diseases known, being equally virulent throughout its course. The poison of the disease has not as yet been demonstrated, although it is known to be given off from the bodies of the sick and the dead, to be carried in the air, and to be retained in bedding, clothes, carpets, etc. for a considerable time, so that the poison is conveyed not only from person to person, but also by clothing and bed-rooms. But a very few persons are exempt if they be sufficiently exposed, and the more prolonged and concentrated the

TYPHUS FEVER.

PLATE 4.

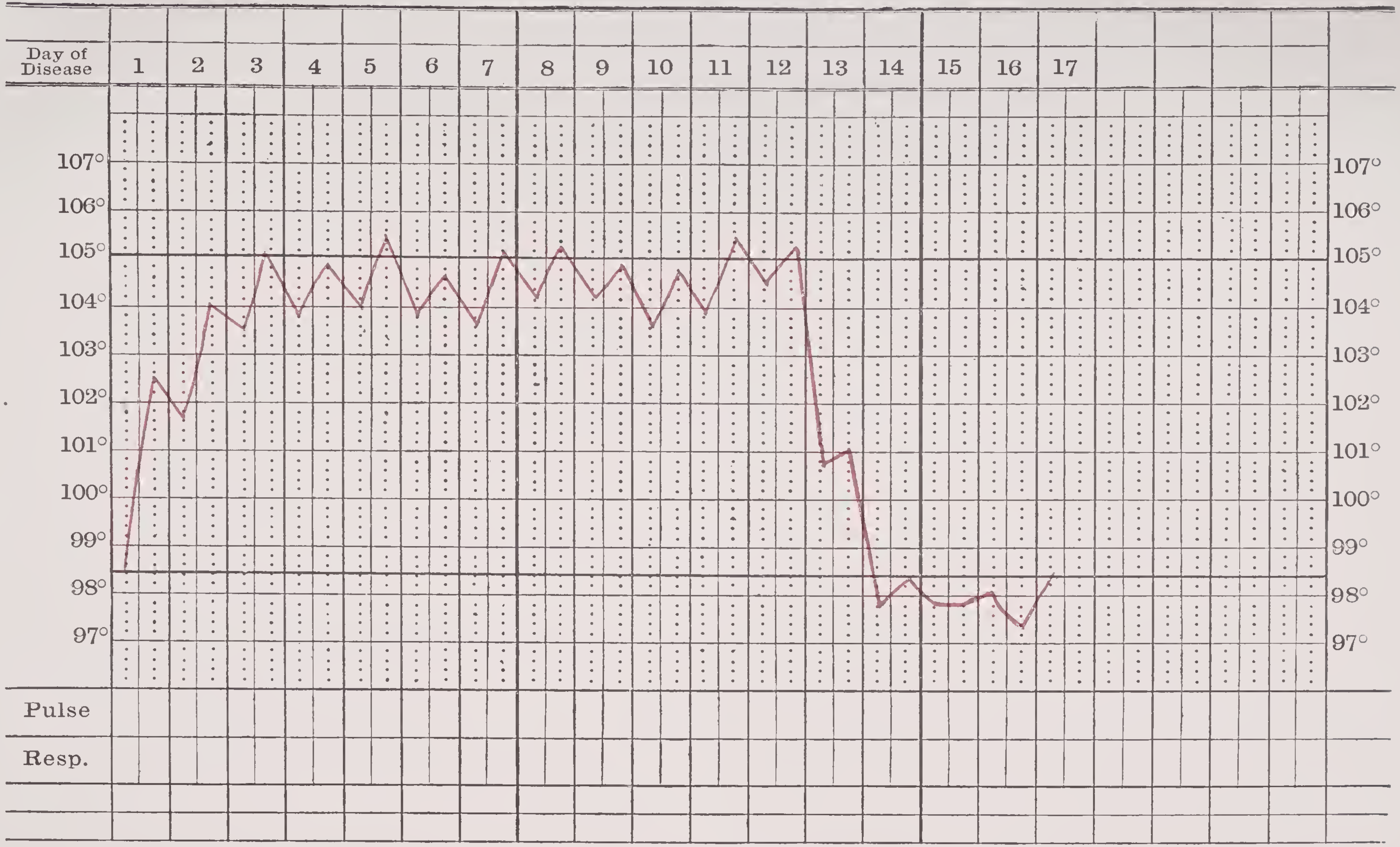


Chart of typhus fever (Pepper).

exposure the more certainly will they be attacked. One attack usually procures immunity. While no age is exempt, the majority of cases occur between the fifteenth and thirtieth years.

Pathology.—The eruption is the only characteristic lesion. After death there may be found a number of morbid conditions common to any of the severe infectious diseases.

Symptoms.—The period of *incubation* is about twelve days, although cases may develop as early as twelve hours or as late as three weeks after exposure.

The *onset* is abrupt, although in some cases it is preceded for a few days by malaise and frontal headache. The initial symptoms are a chill, a rise of temperature, headache, and prostration. The chill, which is usually sharp and severe, may be repeated.

The temperature rises suddenly, attaining its maximum from the third to the fifth day, reaching 103° F. in mild and 105° F. in severe cases. During the first week the temperature remains steadily continuous, becoming somewhat higher in the second week, but with morning remissions. In bad cases there may be hyperpyrexia. From the twelfth to the fourteenth day comes the crisis, the temperature falling rapidly—sometimes a drop of 4° to 5° F. in a few hours, although usually it takes twenty-four to forty-eight hours before the temperature reaches the normal; this fall of temperature is accompanied by an improvement of all the symptoms, the patient entering at once upon the period of convalescence.

The headache is usually frontal and is very severe—more so than in any other disease except meningitis. With the headache are pains in the back and bones and soreness of the muscles.

Prostration is marked from the onset, the patient taking to bed from weakness within a few hours. It may be so severe and sudden that the patient will fall in the street without previous warning.

Nausea and vomiting are commonly present, and may be distressing. The tongue is heavily coated in the first week, later having a tendency to become dry and brown, with sordes on the teeth and gums. The bowels are usually constipated, although there may be diarrhœa.

The pulse is rapid and full, being between 100 and 120; it becomes more rapid and feeble during the second week. Marked slowness of the pulse may be observed at any time of the disease and may continue into convalescence. A drop in the pulse without improvement in other symptoms is not a good sign. The urine usually shows the presence of albumin and casts in moderate amounts from an acute degeneration of the kidney. More rarely there are present the urinary changes of acute exudative nephritis.

Early in the disease there is a look about the patient which is often of diagnostic value. The face assumes a dusky flush, the conjunctivæ are injected, and the expression is dull and vacant, the whole appearance suggesting marked intoxication. The pupils are contracted.

The characteristic eruption appears from the third to the fifth day, although it may be seen as early as the second and as late as the seventh day. It consists of small, irregularly rounded spots, of a dirty-pink color, appearing first on the abdomen and chest, and becoming more general, although rarely seen on the face. It appears in one crop, and is all out in from two to five days, lasting from seven to ten days and then slowly fading. It is usually abundant, though in some cases it may be scanty. At first the spots are slightly elevated and disappear on pressure, but after several days they become petechial and more permanent, remaining after pressure. They have no well-defined margin. In children the eruption resembles that of measles, and from the mottled appearance given by it to the skin the eruption has been termed the *mulberry* rash. In some cases there is added a diffused, deep mottling of the skin with large purplish blotches; in others there are hemorrhagic spots or a general erythema. These manifestations are not characteristic and are inconstant.

Cerebral symptoms are marked and appear early. The headache, so marked at the onset, usually becomes masked by other nervous symptoms by the end of the first week. Delirium is a fairly constant symptom. In very severe cases it may come on in the first twenty-four hours of the disease in the form of an acute mania. In less severe cases it is not seen until the end of the first week. It may then be only a mild delirium at night, or it may be more decided, persisting throughout the day. This latter form is often associated with delusions which at any time may render the patient violent. In severe cases, during the second week there may be observed alternately with the delirium a form of deep stupor known as "coma vigil," in which the eyes are wide open but the patient is unconscious.

Deafness may appear in the second week without assignable cause, but from it the patient usually recovers.

If the case is to end fatally, the temperature rises, often to 106° or 108° F. before death, the delirium and stupor become more decided, there may be retention of urine and incontinence of feces, the pulse becomes more rapid and feeble, and death occurs from exhaustion from the toxæmia. Should the patient survive until the third week, death usually results from a complicating pneumonia.

If the case is to recover, on about the fourteenth day there is a decided fall in the temperature, the patient frequently falling into a refreshing sleep from which he awakes weak but convalescent. In some cases this crisis occurs as early as the seventh day, or at this time there may be a decided remission in the temperature, practically an abortive crisis, which is to be considered a favorable omen. In other cases the crisis may be deferred as late as the twenty-first day.

In some epidemics light cases are seen, running a mild course, with a temperature usually under 102° F., with but moderate cerebral symptoms. The crisis usually occurs between the seventh and the twelfth day. Occasionally there are observed in severe epidemics malignant cases in which the patient is overwhelmed by the virulence of the disease. There are rapidly developed a sudden temperature, usually high, progressive heart failure, stupor, and coma, and death may result in from twelve to twenty-four hours or within two or three days. In these cases no regular eruption is seen, but ecchymoses and hemorrhagic spots are irregularly developed.

Complications.—There may be broncho-pneumonia, which in rare cases is complicated by gangrene of the lung. Gangrene of the extremities or cancrum oris in children has been observed. Meningitis is rare and is always fatal. Abscesses in the skin and the joints may occur, and suppurative parotitis is not uncommon. There may be hemorrhages into the skin or from any of the mucous membranes accompanying serious cases. Thrombosis of large veins or of cerebral sinuses may occur.

Convalescence is usually rapid at first, although it is months before it is complete. There are no relapses. The patient may be left dull and feeble-minded, from which condition the recovery is gradual. A few patients develop acute mania in convalescence, but the ultimate prognosis is generally good. Paralysis from post-febrile neuritis is not uncommon.

Prognosis.—The mortality is from 10 to 20 per cent., varying with the nature of the epidemic, the previous condition, and the age of the patient. The disease is rarely fatal in young subjects, but is very serious in those past adult life. Complicating inflammations alter the prognosis according to their nature.

Treatment.—The patient should be isolated thoroughly from the start. To lessen the danger of contagion to nurse and to physician, the windows must be opened freely to admit fresh air. If possible, the patient should occupy two rooms, one by day and one by night, the freest ventilation thus being afforded. In epidemics the cases are best treated in tents, the patients being protected in winter by extra bedding. This fresh-air treatment is not only a prophylactic measure, but seems also to lessen the actual mortality. Windows should be protected by bars in case mania develops.

There is no specific treatment for the disease. Formerly mineral acids were so considered, but they are given now only because they afford a pleasant acidulated drink and do no harm.

The treatment, then, is entirely symptomatic. The temperature is best treated by hydrotherapy, as in typhoid fever, the bath being given as soon as the temperature reaches 103° F. Internal antipyretics should be avoided if possible, because of their depressing effect. Alcohol in some form is demanded in almost all cases, and it may be given in large doses until a good effect is observed on the heart's action: 10 to 20 ounces of whiskey may be required in the twenty-four hours. The delirium and headache should be treated by sedatives combined with hydrotherapy. Other symptoms should be treated on general principles.

RELAPSING FEVER.

Definition and Synonyms.—Relapsing fever is an acute infectious, contagious disease due to a spirillum, and characterized by a febrile paroxysm of about six days' duration, followed usually by one or more similar recurrences at regular intervals. *Synonyms:* Famine fever; Spirillum fever; Relapsing typhus.

Etiology.—The disease is a rare one, occurring in epidemics which last but a short time and then die out, leaving, except in very rare exceptions, no endemic cases. Epidemics were seen in Philadelphia in 1844, and in New York and Philadelphia in 1847 and 1869. The last epidemic of any importance was in Russia in 1886.

Epidemics have frequently been associated with those of typhus fever, the spread of both diseases being favored by filth, by famine, and by overcrowding of people. Neither age, sex, nor climate exerts any influence upon the epidemics.

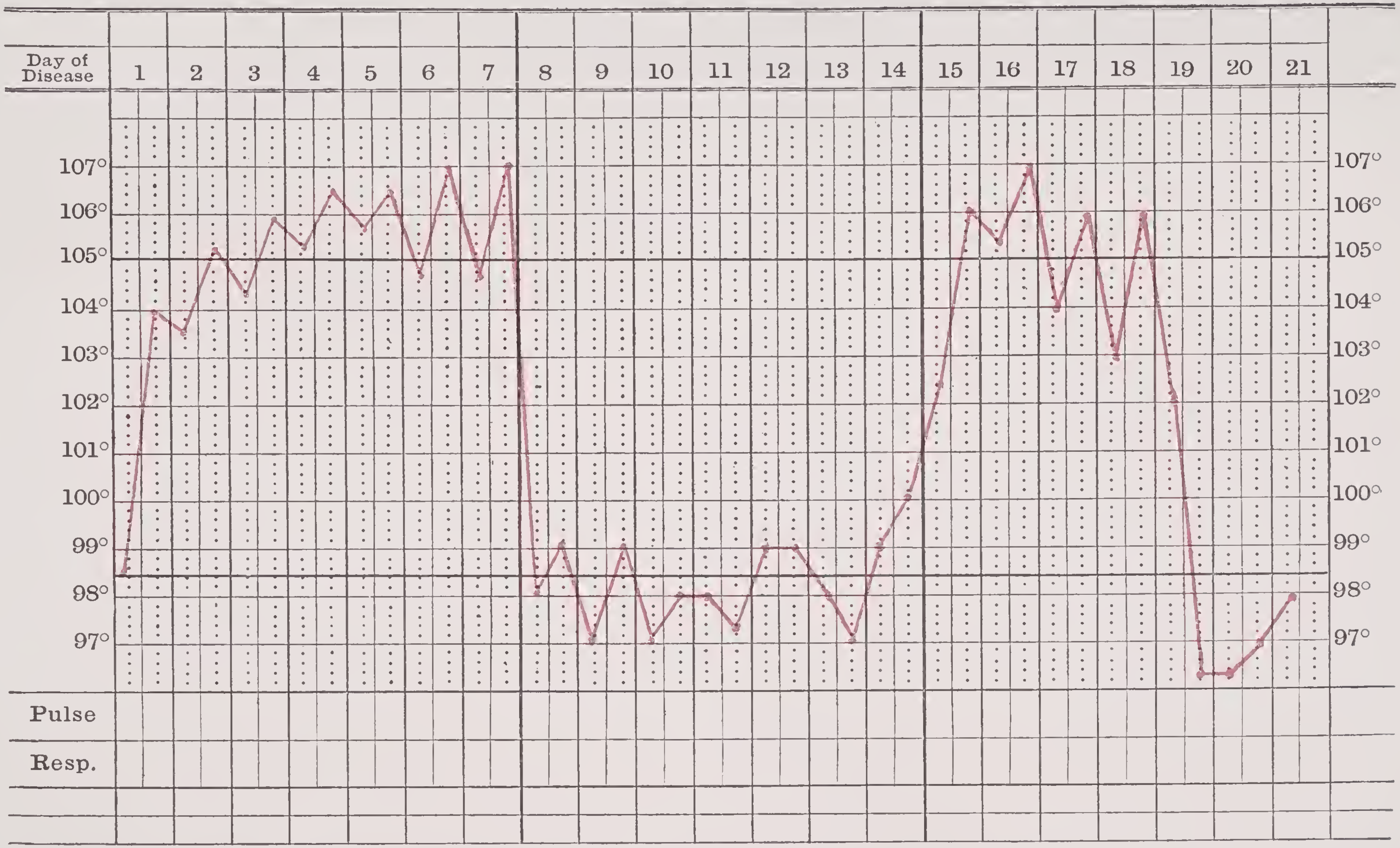
The disease is actively contagious, not only by personal contact, but also through clothes and bedding and through the medium of a third person. One attack does not secure immunity from subsequent attacks.

The exciting cause is infection by a spirillum or spirochæte, first described in 1873 by Obermeier. The spirillum is a slender spiral filament endowed with motion, its length being three to six times as long as the diameter of a red blood-cell. It is present in the blood, but only during the febrile paroxysm. Before the crisis and in the intervals between the paroxysms only round glistening bodies are seen, which bodies are supposed to be the spores. Inoculations of the spirillum into man and monkeys have reproduced the disease.

Pathology.—There are no lesions characteristic of the disease. The spleen is large and soft and may rupture. There are parenchymatous changes in the liver, the kidneys, and the heart-muscle. There may be internal hemorrhages. The tissues may be jaundiced.

PLATE 6.

RELAPSING FEVER.



Typical case of relapsing fever (Pepper).

Symptoms.—Incubation may be short, the attack developing a few hours after exposure. Ordinarily the period is from five to seven days, during which time there are no symptoms. The disease begins abruptly with a chill, followed by a rapid rise of temperature, running to 103° to 107° F. within twenty-four hours. There are severe and distressing pains in the head, back, and bones. Prostration is well marked at the onset, the patient taking to his bed at once. The pulse is full and varies between 110 and 130. There is usually nausea; there may be severe vomiting, and in some cases vomiting of blood. In severe cases there may be delirium. Convulsions may be the first symptom in young subjects.

The liver and spleen are regularly enlarged and tender. Blood-examination shows the spirilla in active movement among the red blood-cells. Jaundice may be present in the attack or in any of the relapses. It belongs to the severer forms of the disease.

There is no characteristic eruption, but in certain cases there may appear small reddish spots somewhat resembling the eruption of typhus fever, or there may be petechial spots. The urine contains a slight amount of albumin and casts from parenchymatous degeneration.

While the symptoms are at their height the paroxysm suddenly ceases, the temperature suddenly falling by crisis, and the patient is left weak but convalescent. This remarkable crisis usually occurs between the fifth and the seventh day, although it may occur as early as the third or as late as the tenth day. This abrupt crisis, which is always a time of danger, may be accompanied by profuse sweating or exhausting diarrhœa. In some cases there may be collapse, or hemorrhages from the stomach, the intestines, or the kidneys.

After such an attack the patient may pass on to recovery, but in the majority of cases, after an interval of about seven days (the limit is from five to twenty days), the patient is again suddenly seized with a repetition of all the symptoms of the first attack, the spirilla again becoming present in the blood. This second attack usually is less prolonged than the first, lasting about three days, although it may last only one day or be prolonged for a week. This attack terminates by crisis as does the first. The patient may now pass on to complete recovery, or there may be a series of these relapses (up to five or six), these subsequent attacks becoming shorter and less severe.

Complications.—There may be hypostatic congestion of the lungs or broncho-pneumonia. Laryngitis and œdema of the glottis may occur. There may be rupture of the spleen with internal hemorrhage and peritonitis. Nephritis is of rare occurrence. In severe cases there may be hemorrhages from the stomach, the intestines, or the kidneys.

Convalescence is apt to be slow and tedious, especially if there has been a series of relapses. There may be paralyses of groups of muscles from post-febrile neuritis. There may be ophthalmia with loss of sight.

The **prognosis** is good. The mortality is from 2 to 4 per cent.

Treatment.—There is no specific treatment. Cases are to be isolated thoroughly, and clothing, etc. disinfected, to prevent the spread of the disease. The treatment is on general principles. It is especially important that the patient should be well watched at the time of crisis for indications of heart failure. The diarrhœa which often occurs at this time should be treated promptly. After the crisis the patient should be kept in bed for at least a week and on fluid food.

SMALL-POX.

Definition and Synonym.—Small-pox is an acute infectious, contagious disease with marked constitutional symptoms and a characteristic eruption. *Synonym:* Variola.

Etiology.—This disease was known to the ancients and was fully described by the older writers. Formerly it occurred in devastating plagues, but since the discovery of vaccination it has become less and less frequent, so that cases of true small-pox are but rarely seen. With universal vaccination the disease bids fair to become extinct.

Small-pox is actively contagious throughout its entire course. The contagion, which is conveyed by personal contact or by the medium of a third person, emanates from the bodies of the sick and the dead, floats in the air, and can remain in bedding, clothes, cabs, and rooms for months and years without losing its virulent properties. It is inoculable by the contents of the vesicles and pustules and by the blood of the sick, and is most actively conveyed by the scales and crusts thrown off by the patients; these scales float in the air as a fine dust, and in this way may be carried long distances.

The exact nature of the contagion has never definitely been determined. Micro-organisms have been found in the contents of the vesicles and pustules, but these micro-organisms are common to all suppurative processes.

Susceptibility to the disease is almost universal except among those protected by vaccination or by a previous attack. No age is exempt, the disease even attacking the fœtus *in utero.* Negroes are more susceptible than other races, though this may be due to their neglect of vaccination. Vaccination properly performed procures immunity, as does a previous attack, although in rare cases the immunity may not extend beyond a term of years (five to fifteen).

The disease exists in nearly all countries as an endemic disease, a certain number of cases occurring every year. Under favoring circumstances it assumes from time to time epidemic proportions. These epidemics are terribly devastating should they occur among aboriginal races where small-pox has not previously existed.

Pathology.—Aside from the eruption there are no characteristic lesions. There may be found the enlarged spleen and the parenchymatous changes in the liver, the kidneys, and the heart-muscle that are present in all severe infectious diseases. Severe cases may reveal internal hemorrhages.

Symptoms.—The period of *incubation* is usually from ten to fourteen days, during which time there are no symptoms.

Five forms of the disease are described: 1. The discrete form; 2. The confluent form; 3. The hemorrhagic form; 4. The malignant form; 5. Varioloid, or small-pox modified by vaccination or by a previous attack.

1. *The Discrete Form.*—The symptoms are described as occurring in two stages.

(*a*) *Stage of Invasion.*—The onset is sudden, being ushered in by a chill which is violent and severe and which may be repeated. The temperature rapidly rises, reaching 103° or 104° F. in twenty-four hours. There is severe headache, usually frontal, which is marked and characteristic, and which lasts throughout this stage with undiminished severity. With the headache there are regularly severe pains in the bones, muscles, and loins. Especially characteristic is an excruciating pain in the sacrum that is present in about half the cases. The severity of the pains gives no clue to the future severity of the attack. While the headache and backache are common to the onset of all severe infections, there is no disease in which they are so marked as in small-pox, and their occurrence should excite appre-

PLATE 7.

SMALL-POX.

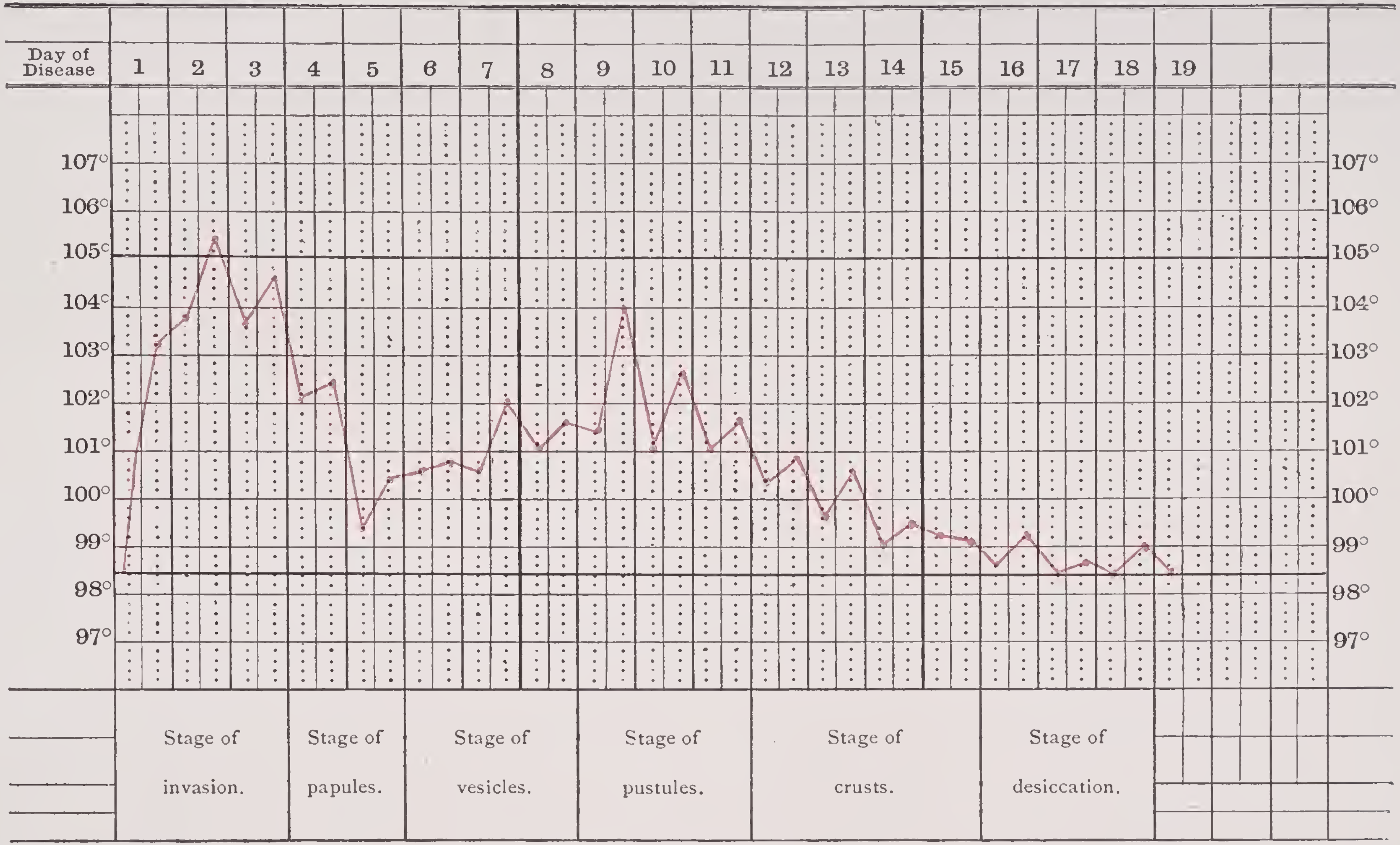


Chart of temperature curve of small-pox (Eichhorst).

hension, especially during an epidemic or occurring in an unprotected patient who has been exposed to the disease.

Vomiting is usually severe and distressing, and prostration is an early symptom, the patient taking to his bed at the very onset. The pulse is rapid and full, increasing in rapidity with the rise of the fever, and becoming weak in proportion to the severity of the disease. Nervous symptoms belong to the severer cases. There may be active delirium, especially in patients with high fever. Convulsions in place of the chill may initiate the disease in children. There are usually considerable restlessness and general apprehension. The eyes are bright; the skin is hot and dry; the spleen is enlarged, and albumin and casts may be found in the urine in small amount.

There is no characteristic eruption in the stage of invasion, although an initial rash may be observed. This rash appears with more frequency in some epidemics than in others, as a rule, however, appearing in from 10 to 15 per cent. of all cases. It consists of an erythema which may be diffuse or macular, closely resembling the eruption either of scarlet fever or of measles, although it differs from both in its localization. It is often associated with small hemorrhagic spots. This initial erythema occurs on the second day, and it appears usually on the hypogastrium, the inner surfaces of the thighs and opposing surfaces of the side of the chest, and the inner side of the arms—regions which are usually exempt from the regular eruption. This early rash lasts but a short time and then fades away.

The stage of invasion lasts regularly three days, during which time the symptoms continue, the temperature usually becoming somewhat higher, so that it may reach 105° to 107° F.

(*b*) *Stage of Eruption.*—The eruption is peculiar in that it passes through successive stages in development, becoming in turn macules, papules, vesicles, and pustules. Beginning on the fourth day of the disease, there appear small round, slightly raised spots, involving first the face, the edge of the hairy scalp, and the backs of the wrists, and extending within twenty-four hours over the trunk, the arms, and, last of all, the legs. On the second day of the eruption the spots become papules which are hard to the feel, suggesting bird-shot imbedded in the skin. By the third day the papules change to vesicles which have each a depressed umbilicated centre without any tendency to rupture. Should these vesicles be pricked, they do not completely discharge their contents. Vesiculation is complete on the sixth day; the vesicles then become pustular. The central umbilication usually disappears, so that there result pustules each with a rounded summit surrounded by a zone or areola of inflamed and swollen skin which burns and itches and causes the feeling of distressing tension. The face is now strikingly swollen and disfigured; the eyelids are closed by œdema. In severe cases the skin between the pustules becomes diffusely inflamed, adding to the distress and disfigurement of the patient. The pustules are completely developed on the eighth or ninth day of the eruption, and after continuing for about three days begin to dry up and to form scabs and crusts which, falling off, leave pigmented spots that persist for months. If the *cutis vera* has been involved by the pustules, permanent depressed white cicatrices must necessarily result. The desiccation requires a week or ten days for its completion, and it is attended by much itching.

There are usually similar eruptions in various mucous membranes, especially those of the mouth, pharynx, nasal cavities, larynx, and trachea, and less frequently of the upper portion of the œsophagus, the bronchi, the conjunctiva, the vagina, and the rectum. When the eruption appears on mucous membranes, it is modified by the heat, moisture, and friction of the parts, and, instead of pustules being formed, the vesicles become macerated and form ulcers, more or less deep, which may become confluent.

As the eruption appears the temperature suddenly falls —not, however, quite to the normal, but still remaining a little high. The constitutional symptoms remit in their severity, especially the headache and backache, so that the patient feels quite comfortable, and often is able to leave the house and to apply for treatment at a hospital or a dispensary.

The amelioration of the symptoms continues until the pustular stage is reached, when there is developed a rise of temperature to 101° to 105° F. with a return of all the symptoms. This fever, which is termed the secondary or suppurative fever, is often remittent. During this stage there may be active delirium, so that the patient may do himself or his attendants bodily injury. The distress during this period from the itching, tension, and burning of the skin is almost unbearable. The patient is disfigured and often is hardly recognizable.

The period of secondary fever lasts for from three to eight days, and then as desiccation is established the temperature gradually falls and convalescence is begun. The hair frequently falls out, and in some cases does not grow again.

2. *The Confluent Form.*—Here the typical symptoms are present in the most complete development. The stage of invasion is short, lasting but two days as a rule. It is also severe, the fever being high, with pronounced headache and frequently with active delirium. There is no period of cessation with the appearance of the eruption, although the symptoms may remit slightly in their severity.

The eruption is profuse, the pustules becoming confluent, so that whole areas, especially of the face and hands, are converted into suppurating blebs. The patient is unrecognizable, the distress is extreme, and the stench is penetrating and intense.

The mucous membranes are usually extensively involved. The suppurative fever is apt to run high and is attended by symptoms of a septic character—irregular chills followed by exacerbations of fever, rapid and feeble pulse, diarrhœa, and extreme prostration with a low muttering delirium which at times may assume an active form. In this septic condition the patient may die. Desiccation continues into the third and fourth weeks, during which time the symptoms of the secondary fever persist with diminishing intensity.

3. *The Hemorrhagic Form.*—This form is characterized by hemorrhages occurring in the pustules and the skin and from any of the free mucous surfaces—from the nose, mouth, lungs, stomach, intestines, kidneys, or uterus. This form of small-pox may occur in weak, debilitated subjects, or the hemorrhages may complicate the severer forms of the disease.

4. *The Malignant Form.*—The invasion in malignant cases is characterized by extreme prostration and enfeebled heart-action, the initial rise of temperature not being high. In from eighteen to thirty-six hours there appears an erythema (resembling scarlet fever with large hemorrhagic blotches) which occurs extensively over the body, especially the abdomen and thighs. There are frequently hemorrhages from the mucous membranes. The regular eruption appears somewhat later, but does not run a perfectly typical course, being represented in some cases only by a few vesicles. In some cases death results in a few hours, before any eruption occurs, the patient being overwhelmed by the virulence of the disease. In other cases death does not occur until from the third to the seventh day.

5. *Varioloid.*—This disease is small-pox occurring in a modified form in a person who is but partially protected by vaccination or by a previous attack. According to the extent of the protection, varioloid occurs in all grades of severity, so that the severer grades merge into true, unmodified variola, while in the lesser grades the patient may not seem to be sick at all. No matter how trifling the attack may be, it is true variola, and is capable of causing the disease in others in even the most malignant forms.

Generally varioloid runs a milder course and one of shorter duration than small-pox. The stage of invasion lasts for two or three days. There may be a chill, with moderate fever, headache, pains in the back and bones, and vomiting, while in very light cases these symptoms may not be severe enough to prevent the patient from being at work and out of doors.

The eruption is that of variola, but it is less abundant, the pustules do not reach the same size nor penetrate so deeply, and the areola of inflamed skin is frequently absent. Many of the vesicles dry up and are not converted into pustules. With the appearance of the eruption the fever falls to normal and the other symptoms of the invasion disappear and frequently do not return. In more severe cases the symptoms of the secondary fever are slight and last but a day or so, so that there is a striking lack of proportion between the appearance of the patient and the degree of his constitutional symptoms. Desiccation begins from the fifth to the seventh day, and the resulting cicatrices are small or are absent.

Complications and Sequelæ of small-pox are numerous, and are due not only to the severe infectious nature of the disease, but also to the suppurative foci. There may be laryngitis, œdema of the glottis, or necrosis of the cartilages, resulting in stenosis, necessitating intubation or tracheotomy.

Broncho-pneumonia is a common complication, especially in the severer cases. There may be pleurisy. Nephritis is rare, although the urine usually contains a small amount of albumin. There may be orchitis. During the suppurative stage there may be developed septicæmia or pyæmia or arthritis. In rare cases a disseminated myelitis has been observed. Convalescence may be interrupted by boils, by gangrene of the skin, or by erysipelas. Ulceration of the cornea is but rarely seen. During convalescence there may be a post-febrile insanity or paralyses from peripheral neuritis.

Prognosis.—The prognosis depends upon the degree of protection afforded the patient by vaccination. Varioloid is very seldom fatal in those protected by vaccination, while in those totally unprotected the mortality ranges from 30 to 40 per cent. The prognosis depends also upon the severity of the attack, the hemorrhagic and malignant forms being almost certainly fatal, the confluent form being very dangerous, while the discrete form affords a large percentage of recoveries. It depends also upon the age and the condition of the patient, being most fatal in children and old people, in the debilitated, and in drunkards. The prognosis is affected also by the presence of complications.

Treatment.—If vaccination and revaccination could be performed thoroughly, variola would become extinct. After exposure to variola, revaccination should be resorted to, and it is probable that vaccination even in the earliest stages of the disease itself, if done before the fourth day, greatly modifies its severity.

Patients with variola should properly be treated in contagious hospitals, as few private houses afford sufficient means for thorough isolation and disinfection. When this cannot be done, quarantine should be conducted on the strictest principles, and should be continued until the skin and the hair are absolutely free from crusts and scales. All articles that come in contact with the patient should be sterilized or be destroyed.

The room should be kept moderately cool and well ventilated. Patients are rendered worse by being kept too hot. The occurrence of mania should be anticipated by careful watching and by having the windows barred. The diet should be of milk.

The fever is best treated on hydropathic principles; the headache and backache are to be combated by opium or phenacetine. Sleep may be procured by sulphonal, chloralamide, or codeia.

The patient should, if possible, be prevented from scratching. Great care should be exercised in the strictest cleansing of the eyes and in preventing them from being infected by their being rubbed with pus-stained hands. For the cleansing a saturated solution of boric acid is to be preferred.

As the extent of the pitting depends entirely upon the depth of the pustules, there is no method of treatment known by which pitting can be prevented. It seems, however, that the intensity of the pustules is modified by excluding the light and by keeping them covered with strips of gauze constantly wet with weak solutions of bichloride or of carbolic acid. This application also relieves the pain and itching. Isolated pustules may be painted twice a day with 10 per cent. carbolic acid in alcohol. When crusts form they are to be softened by applications of lard or of vaseline to prevent them from floating in the air and thus carrying the infection. The other symptoms and complications should be treated on general principles.

VACCINIA; VACCINATION.

Vaccination was first performed in 1798 by Jenner, and its value is now universally acknowledged. The vaccine may be procured by means of the scabs of patients vaccinated (humanized virus), or be taken direct from the calf (bovine virus). If the humanized virus be used, it is of the utmost importance that it be taken from a healthy subject free from every trace of syphilitic and tubercular taint. As a rule, bovine virus obtained fresh from reliable sources should be used.

In a primary vaccination there appears in from twenty-four to thirty-six hours a papule which on the fifth or sixth day becomes an umbilicated vesicle surrounded by an indurated inflammatory zone. This papule on the eighth or ninth day changes to a pustule, which dries up, and the brownish scab resulting falls off on the twentieth to the twenty-fifth day, leaving the characteristic scar. In patients who have already been vaccinated successfully there may be either no result, or an irregular atypical vesicle, or a local ulceration on an inflammatory base.

About the third day of vaccination there begins a moderate fever, often preceded by chilly feelings, with malaise, restlessness, and irritability, these symptoms being especially marked in children. These symptoms increase moderately until pustulation is completed; then they subside. The neighboring lymphatic glands become swollen, painful, and tender.

The protection afforded varies with the completeness of the vaccination and with the time that has elapsed since it was last performed. Every baby should be vaccinated, preferably between the second and third months, and thereafter every seven years—oftener, however, if exposed to small-pox or during an epidemic. Should, under these latter circumstances, a revaccination be unsuccessful, it should be repeated.

Complications of vaccination are due either to lack of cleanliness or to impure virus employed, and should not occur if proper precautions be taken. There may be sloughing ulcers, gangrene of the skin, or erysipelas. Septicæmia may develop in neglected cases. Impetigo contagiosa has been known to result. The patient may be inoculated with syphilis from humanized virus taken from diseased patients, the double infection resulting in the primary lesion at the site of inoculation. There may be observed vesicles about the inoculation, or a general eruption of vesicles from absorption of the virus.

VARICELLA.

Definition and Synonym.—Varicella is a contagious disease especially of childhood, and is characterized by a vesicular eruption. *Synonym:* Chicken-pox.

Etiology.—Varicella occurs in sporadic, endemic, and epidemic forms. It is contagious throughout its course, but the contagious principle is of a low grade of intensity. It is almost exclusively a disease of children, but it may occur in young adults. One attack does not afford absolute immunity from subsequent attacks.

The exact virus has not been determined definitely. There is no connection, immediate or remote, between this disease and variola or varioloid.

Symptoms.—The period of incubation is from eight to seventeen days, and it is unattended by symptoms. The invasion is marked by chilly feelings in some patients and by feverishness, the temperature rarely being higher than 102° F. except in very young children, in whom a fever of 104° F. is not unusual Convulsions are rare at the onset. There is apt to be vomiting, and the child complains of lassitude and of pains in the back and legs. These symptoms last for but a few days. In some cases the constitutional symptoms are so slight that except for the eruption the child may seem well.

The eruption appears in about twenty-four hours, first on the chest and back or on the forehead and face, and consists of small raised spots which in a few hours become vesicles. These vesicles vary greatly in size, the larger ones usually appearing on the forehead; they are rarely umbilicated; they collapse, as a rule, by a single puncture; they appear superficial, and not deeply seated; and they are but rarely surrounded by an inflammatory halo. A few vesicles become pustular, but the majority in a day or so simply shrivel up, leaving crusts which fall off, usually leaving no cicatrices, although circular scars may result from the larger and deeper vesicles. Fresh crops of eruption appear during the first two or three days, so that the eruption can be seen in all stages of development. The eruption also appears in any of the mucous membranes; the vesicles, becoming macerated, leave superficial ulcers.

In a few cases there appears a scarlatinal rash before the regular eruption. In rare cases, in debilitated children, there may be gangrene of the skin, or hemorrhages into the eruption and skin and from mucous membranes.

The **prognosis** is regularly good except for the severer forms in very puny, weakly children.

Treatment.—The patient should be kept in bed during the febrile stage, and in the house until the skin is free and clear. A child should be isolated from other children, but may associate with adults.

SCARLET FEVER.

Definition and Synonym.—Scarlet fever is an acute infectious, contagious disease with an eruption, fever, and pharyngitis, and with a tendency to cause an inflammation of the kidneys. *Synonym:* Scarlatina.

Etiology.—The disease is contagious from the invasion until the end of desquamation. The poison is conveyed by the exhalations of the patient, by personal contact, or through the medium of a third person, and is spread by the scales of the desquamation; these scales, which float in the air and are deposited in clothing, bedding, and rooms, may remain in the hair long after convalescence. The poison possesses extraordinary vitality, so that contaminated fomites may convey the contagion even after months or years. The poison may be carried great distances and may be conveyed also in milk. The exact nature of the poison has not definitely been determined.

The disease occurs both in isolated cases, due always to contagion of some previous case, and in epidemics, which vary greatly in their virulence. It is most common in the fall and winter months.

Susceptibility to the disease varies, some people being exempt. This exemption may run in families. The disease usually attacks children, 50 per cent. of all cases occurring before the fifth year, 90 per cent. before the tenth year. Young infants are rarely attacked. Adults become less and less susceptible with advancing years. One attack, with but the rarest exception, procures future immunity.

The disease is said to occur with special frequency in puerperal women and after surgical operations, but it is now supposed that these cases are really septicæmia with a septic erythema, which often desquamates, as does any long-continued hyperæmia of the skin.

The relation between scarlet fever and diphtheria is at present unsettled. A patient may have the two diseases together from a mixed infection, or a scarlatina may occur with membrane in the throat, or a diphtheria may occur with a septic erythema. Careful bacterial investigations are required to settle the relationship of the two diseases.

Pathology.—The characteristic lesions consist in the eruption and the pharyngitis. No traces of the eruption are observed after death, except in the hemorrhagic form. The pharynx usually presents the appearance of a catarrhal inflammation.

The complicating inflammations are exceedingly numerous and important. There may be follicular tonsillitis or pseudo-membranous inflammation of the tonsils, pharynx, or larynx. There may be cellulitis of the neck that may go on to gangrene or suppuration, the pus containing streptococci. There may be infection of the middle ear with otitis media, perforation, and deafness.

Respiratory complications are not common, although broncho-pneumonia may occur. In rare cases there may be pericarditis or endocarditis, and less frequently meningitis. The spleen is usually found enlarged during the height of the disease.

In nearly all cases there is found a parenchymatous degeneration of the kidney, but there may be either acute exudative or acute diffuse nephritis. These renal changes are the most important and formidable of all the complications of the disease, constituting the so-called "scarlatinal nephritis."

Symptoms.—*Incubation.*—This period is pretty constantly seven days, although its extreme limits are between twenty-four hours and two weeks. During this period there are no symptoms.

Invasion.—The invasion is sudden and striking, being marked by fever, sore throat, and vomiting. This trinity of symptoms occurring suddenly should always excite the suspicion of scarlatina. There may be initial chilly feelings, or convulsions in young children. An actual chill is rare.

The vomiting, which is usually severe and apparently uncalled for, may be projectile. The fever mounts rapidly, and reaches 103° to 105° F. within a few hours, few diseases showing such a rapid rise. There is pain on swallowing; the throat is dryish; the glands at the angle of the jaw are swollen and tender. On inspection there may be only the appearance of a catarrhal pharyngitis either mild or severe, or there may be added a follicular tonsillitis. The pulse becomes rapid and is usually of high tension. The face is flushed, and the child at once looks seriously ill. There may be repeated convulsions or talkative delirium, according to the severity of the disease and the nervous constitution of the patient. There is usually some nocturnal wandering of the mind. In rare cases either the vomiting, the fever, or the sore throat may be absent.

Eruption.—The eruption appears on the evening of the first or on the second day, twelve or twenty-four hours after the onset, although it may be delayed until forty-eight hours; it consists of minute red points, not elevated above the level of the skin, appearing first on the throat, breast, and back, and rapidly spreading over the entire body, imparting to it the color of a boiled lobster. The chin and the mouth are usually left clear, giving a highly characteristic appearance and making diagnosis easy between this disease and measles or small-pox. The face, moreover, is not usually so much involved as in measles. By stroking the rash white lines stand out with characteristic vividness. The eruption may cover the entire body, or it may appear in patches separated by areas of normal skin, giving a peculiar mottled appearance, or in rare cases the eruption may appear only on the face, the body, or the extremities.

There is usually considerable itching, especially during the intensity of the eruption. When the eruption is well marked the skin is often diffusely swollen and inflamed, producing an uncomfortable feeling of tension. The rash lasts for two or three days and then gradually fades.

While the eruption is developing the pharyngeal symptoms continue. After the initial vomiting of the onset there are rarely gastro-intestinal symptoms. The tongue presents the so-called "strawberry" appearance, the papillæ in the tip and edges standing out like shining red pearls above the epithelial coating. The fever remains high during the development of the eruption, frequently reaching as high as 105° or 106° F., and then gradually subsides with the fading of the eruption, becoming normal about the seventh day, although there is frequently a slight afternoon rise throughout the entire period of desquamation. At the height of the fever there are apt to be nervous symptoms—restlessness, nocturnal wanderings, or even delirium with convulsions—but with its subsidence these symptoms rapidly improve, the pharnygitis disappears, and convalescence begins.

Desquamation.—Desquamation begins, after the fading of the eruption, in large flakes. This desquamation is especially noticeable about the hands and feet, epidermal casts of the fingers and toes being frequently thrown off entire. These flakes are potent factors in the spread of the disease, and the patient should not return to ordinary life until desquamation is completed. Usually desquamation lasts into the third or fourth week, but it may last much longer. It is exceedingly rare for it to be absent altogether.

Variations from the regular course of scarlatina are frequently observed, and are due in most part to varying intensity of infection. Of these variations, mention will be made only of the most common.

1. *Mild and Rudimentary Cases.*—The invasion is marked only by a slight transitory fever and moderate angina. The eruption may be scanty, extending over a small portion of the body only, disappearing in from a few hours to a

day, or it may be absent altogether. There is usually, following the eruption, a slight though evident desquamation lasting into the second or third week. These cases are frequently spoken of as "sympathetic sore throats," and are usually seen in partially unsusceptible adults who have been exposed to scarlatina. They are really true cases of mild scarlatina, and are not only capable of spreading the disease, but may also be followed by complications, especially nephritis. To such a rudimentary scarlatina many a supposedly primary nephritis owes its origin.

2. *Severe and Prolonged Cases.*—The onset is usually severe, the temperature rising to 106° or 107° F. and the prostration and cerebral symptoms being pronounced. The eruption in these cases may be scanty. The fever and the constitutional symptoms usually continue into the second week. These are bad cases, but they are not to be considered as hopeless.

3. *Malignant Cases.*—The invasion is marked by a rapid rise of temperature, frequently as high as 106° or 108° F., with cerebral symptoms of ominous gravity—restlessness, delirium, convulsions, passing into coma. There is urgent dyspnœa; the pulse becomes increasingly rapid and feeble. In these cases the patient is overwhelmed by the virulence of the poison, and dies in coma or collapse in from eighteen to thirty-six hours. As a rule, there is no eruption, although a scanty atypical rash may appear should the patient live to the second or third day.

4. *Hemorrhagic Cases.*—These are severe cases with a disposition to bleed from the mucous membranes and into the skin, the latter hemorrhages appearing as petechiæ and large ecchymoses. Cerebral symptoms are apt to be pronounced. There are usually vomiting and diarrhœa, and frequently dyspnœa. The temperature may not be high.

The prognosis is bad, the patient usually dying in two or three days.

5. *Angina Cases.*—This type of scarlet fever is characterized by the predominance of throat symptoms and complicating inflammations. The pharynx and fauces are usually intensely inflamed and their tissues much swollen, making speech and swallowing painful and difficult. There is frequently a membranous exudate which may spread from the pharynx and tonsils to involve the naso-pharynx and nose, or downward to the larynx, where the membrane may cause obstruction to the breathing and asphyxia. There may be necrosis of the throat-tissues that may lead to large sloughs, and if large arteries are involved fatal hemorrhage may result. The glands of the neck become enlarged, and frequently proceed to abscesses and cellulitis. Otitis media is apt to result by infection through the Eustachian canals. The patient rapidly passes into profound sepsis with its attendant symptoms—a condition which is rarely recovered from.

Complications.—1. Sufficient mention has already been made of the important *throat and ear complications* and of the glandular swelling and suppurations.

2. The *complicating lesions of the kidneys* are exceedingly common. Very few cases of scarlatina run their course without the kidneys being involved in one of three ways: (1) by acute degeneration; (2) by acute exudative nephritis; (3) by acute diffuse nephritis. These varieties of nephritis have been admirably differentiated by Delafield, and his classification will be followed.

(*a*) *Acute degeneration of the kidney*, or parenchymatous nephritis, belongs to the first and second weeks of the disease, and is not different from the degeneration occurring in the course of any severe infectious disease. The urine may be diminished slightly in quantity, and it usually contains albumin and casts in moderate amounts. The course is mild, unattended by constitutional symptoms, and ends in recovery.

(*b*) *Acute exudative nephritis* belongs to the second and third weeks, following either a mild or a severe attack of scarlatina. In *severe* cases the urine is scanty or suppressed; its gravity usually is unchanged; albumin and casts are abundant; there may be blood. There are uræmic symptoms—headache, nausea and vomiting, dyspnœa, convulsive twitchings. In some cases there is added contraction of the arteries with high-tension pulse and disturbed heart-action. The patient becomes anæmic and the face puffy, the œdema frequently becoming general with fluid in the serous cavities. The temperature is raised, and it remains remittingly high during the acute stage of the nephritis. In *mild* cases there may be only moderate changes in the urine; uræmic symptoms may be unobserved. The fever is slight or absent. Between these two there are all grades of severity. These cases run about four weeks and usually terminate in recovery, a small proportion only being fatal.

(*c*) *Acute diffuse nephritis* occurs in the third week and during convalescence. It is really a post-scarlatinal nephritis, and may develop after either mild or severe cases. It runs an acute or a subacute course. The acute cases begin suddenly and resemble a severe attack of exudative nephritis. The subacute cases develop gradually. There is apt to be repeated vomiting, which is always to be regarded with suspicion in a patient convalescing from scarlatina. Anæmia and dropsy progress slowly. The urine is regularly diminished in quantity, and it contains abundant albumin and casts. In some cases the primary changes are the first symptom. In all cases of diffuse nephritis the disease is apt to continue with more or less rapidity, with or without remission, until the death of the patient. In rarer cases the lesion becomes chronic.

3. *Inflammation of serous membranes*, meningitis, may occur. Endocarditis and pericarditis are more frequent. The so-called "scarlatinal rheumatism" occurs usually as the eruption is fading, and involves especially the hands and feet, although the large joints may be affected as well. The joints are painful, and may be swollen and inflamed. The process is septic, and not rheumatic, in its nature. Recovery is effected in a few weeks, though suppuration or permanent deformity may result.

4. *Inflammation of the muscles*, myositis, may occur, especially in the neck-muscles, with pain, tenderness, and contraction. The muscles may in rare cases remain permanently contracted.

The **prognosis** is influenced by the character of the epidemic (5 to 30 per cent. mortality), the age and general condition of the child, the severity of the attack, and the nature of the complications. A guarded prognosis must always be given, as dangerous complications, especially nephritis, may occur even during convalescence.

Treatment.—The patient should be isolated strictly until the completion of desquamation. The room should be well ventilated, free from draughts, and should have a temperature of about 70° F. The patient should be kept in bed until the temperature has been normal for a week, after which time the child may be allowed about the room. The diet should be of milk during the febrile period; broths, eggs, fruit, and light cereals may be allowed during early convalescence; but no animal food should be taken until the fourth week. The best preventive of nephritis is a rigorous milk diet.

There is no specific medication. Symptoms must be treated on general principles.

Fever.—At the onset aconite may be given in drop doses every quarter of an hour until arterial tension is decreased, and then every two or three hours to hold the pulse at that point. Should the fever be high, it is best treated by hydrotherapy, which has the additional advantage of calming the nervous symptoms. For this purpose baths, the cold pack, sponging with water, or the ice-cap may be employed without the least danger. The "driving in" of the eruption, with disastrous consequences, is mythical. Internal antipyretics should not, as a rule, be employed.

The nervous symptoms may be controlled by hydrotherapy, by phenacetine (gr. ij–v) to a child five years of age, or by chloral. The following prescription has been proven useful:

℞. Chloral hydrate,	gr. ij;
Camphor-water,	♏xv;
Syrup of orange-peel,	ad ʒj.—M.

Sig. Dose for a child four years old, repeated every three or four hours.

Or,

℞. Chloral hydrate,	gr. ij;
Peppermint-water,	℥ss.—M.

Sig. Dose for a child four years old, every three or four hours.

This prescription is also valuable to correct the initial vomiting.

The *pharyngitis* should always be treated. Gargles do not prove of much use in children, and rarely in adults. Potassium chlorate is to be avoided because of its toxic effects on the kidneys. The pain is best relieved by cloths wrung out of hot water, applied frequently to the neck and covered by oil silk. In some cases cold applications to the throat are more grateful. The mouth and throat must be kept clean to avoid the spread of the infection, they being frequently rinsed out with a saturated solution of boric acid, and sprayed every two to four hours with a solution of bichloride of mercury (1 : 5000) or with peroxide of hydrogen (3 per cent. solution).

The daily anointing of the skin with a bland antiseptic ointment relieves the itching and the feeling of tension and prevents the dissemination of the scales. Lard, cacao butter, or olive oil may be used, to each ounce of which carbolic acid (gr. xx), thymol (gr. x), or menthol (gr. x) may be added. Previous to the inunction the skin should be cleansed daily with soap and warm water.

The temperature should be taken at times during convalescence. The heart, lungs, and urine should be examined every few days, even if the progress of the case seems satisfactory.

Nephritis is to be treated as though it were a primary disease.

The septic inflammation of the joints is best treated by hot applications and by saline laxatives. Ichthyol ointment (ʒss to ℥j) may be serviceable. Salicylic acid and its derivatives occasionally seem to do good, but they are uncertain.

Cervical adenitis and cellulitis are to be treated on general rules.

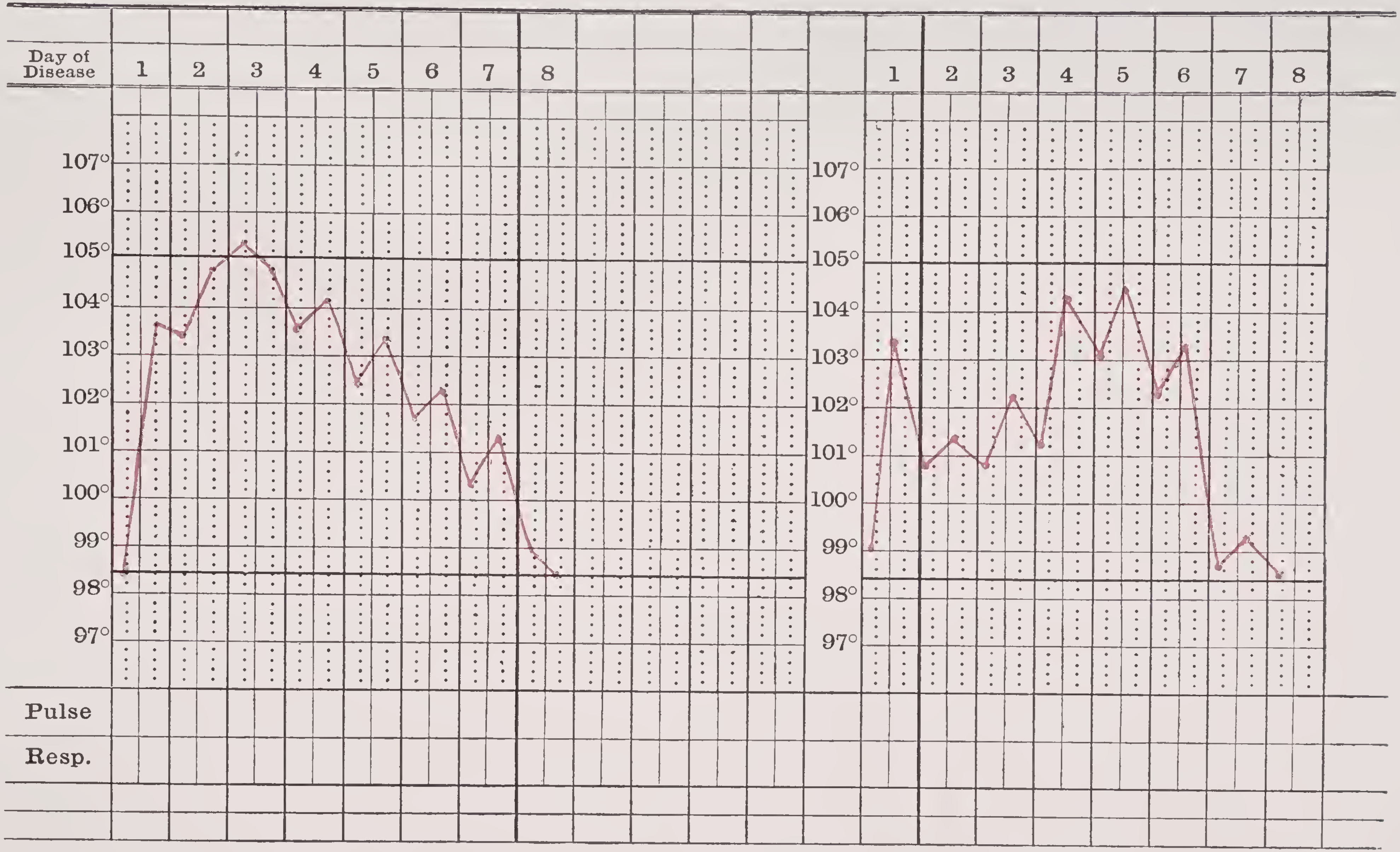
Before the patient returns to ordinary life desquamation must be over entirely. Several warm cleansing baths with repeated shampooing are to be employed to remove all scales from the skin and the hair.

MEASLES.

Definition and Synonym.—Measles is an acute contagious disease characterized by an initial coryza and a maculopapular eruption. *Synonym:* Rubeola.

Etiology.—The disease is endemic, and from time to time assumes epidemic proportions. It most frequently attacks

SCARLET FEVER.—MEASLES.



1. Scarlet fever.

2. Measles.

children, especially those under eight years of age, but unprotected adults are more liable to the infection than to scarlet fever, the infection being more intense and susceptibility to it being much more universal. It is contagious throughout its course, especially during the eruption, and there are grounds for believing it contagious even during the period of incubation. The poison is conveyed by the breath, by the secretions, especially of the nose, and by the branny desquamation. It may be carried by the air, by fomites, or by a third person. One attack does not always procure immunity, but there may be a second, a third, or even a fourth attack, although recurrences are not so common as supposed.

The exact contagion has not been determined, but it is known to possess less vitality and a shorter duration of life than the poison of scarlet fever. Under ordinary circumstances it is usually a mild disease, but epidemics in crowded tenement-houses and in armies may become serious, while epidemics occurring for the first time in savage tribes may be exceedingly fatal, the mortality being even 25 per cent. of the entire population.

Pathology.—The eruption and the catarrhal inflammations of the conjunctivæ and the upper respiratory tract constitute the essential lesions. As complicating lesions there may be found broncho-pneumonia, capillary bronchitis, swelling of the bronchial glands, and less frequently pleurisy or lobar pneumonia. There may be hyperæmia of the gastro-intestinal mucosa with swelling of Peyer's glands.

Symptoms.—The period of incubation is from ten to fourteen days, rarely so long as twenty days. During this time there are usually no symptoms, although in some cases the child may be feverish and irritable.

Invasion.—The child becomes listless and exhibits the symptoms of a feverish cold. There are usually shivering attacks, but a regular chill or an initial convulsion is rare. There are redness of and running from the eyes, with photophobia, sneezing, snuffling, and running from the nose, followed by cough and hoarseness. Sore throat is sometimes complained of, but is not so common nor so severe as in scarlet fever. Examination may show hyperæmia with small red spots on the hard and the soft palate. The temperature rapidly rises to 102° or 104° F., remitting somewhat on the second and third days, becoming again elevated upon the appearance of the eruption. There may be nausea or vomiting and nervous symptoms depending on the severity of the attack and the constitution of the child. In some cases these symptoms are slight, the child not feeling well, having apparently a trifling cold with but moderate feverishness. The stage of invasion lasts for three or four days.

The Stage of Eruption.—The eruption usually appears on the fourth day, being seen first on the face and neck, and then rapidly spreading over the chest and body. It consists of small round spots, slightly elevated, so that they may impart a shotty feeling to the finger; these spots increase in size and assume a roundish or crescentic outline. The rash is hyperæmic, disappearing on pressure, although in some cases there may be petechiæ. The rapidity of development varies, the eruption becoming characteristic in some cases in a few hours, in other cases not for several days. The eruption closely resembles that of poisoning by shellfish or that produced by antipyrine. The eruption is fully developed by the end of two or three days, and then begins to fade, being followed by a fine branny desquamation. At the height of the eruption there may be some swelling and inflammation of the intervening skin, but usually it is normal in appearance.

When the eruption appears the temperature again rises, reaching its maximum at the time of the greatest development of the eruption, after which, usually in the second day of the eruption, the temperature begins to fall, frequently by crisis. Restlessness, sleeplessness, or even general convulsions and delirium, may accompany the exacerbation of the fever. The catarrhal symptoms attain their maximum while the eruption is developing, and then gradually subside.

Variations in its Course.—1. In rare cases there may be no catarrhal symptoms during the period of invasion.

2. There are very light cases in which there may be no eruption. After the regular period of incubation the patient becomes indisposed, feverish, and has a coryza—as it is said, "sickens for the disease," but the eruption is not developed.

3. In some cases the eruption appears as early as thirty-six hours, or it may be deferred until the sixth day.

4. There are cases of marked severity characterized by high fever (105° or 106° F.) and cerebral symptoms, convulsions, delirium, and stupor.

5. In some epidemics, especially in armies and in savage tribes where measles appears for the first time, may appear malignant cases, the so-called "hemorrhagic" or "black" measles. The invasion is sudden and intense; prostration is extreme; there are convulsions or delirium or even coma. The eruption becomes hemorrhagic; hemorrhages occur in the skin and from mucous membranes, These cases are almost always fatal.

Complications and Sequelæ.—The most important are those of the respiratory system. A mild form of bronchitis is common to the disease, but in debilitated subjects, in asylum children, and in severe forms of the disease the inflammation is apt to extend and to lead to broncho-pneumonia. This complication runs a regular course, and is the cause of death in the vast majority of fatal eases. There may be a swelling of the bronchial glands that render them liable to tubercular infections, which may be the origin of acute miliary tuberculosis. Thus in asylums fatal cases of tuberculosis frequently follow epidemics of measles after a little interval. In some cases there may be a tubercular broncho-pneumonia at the start from added tubercular infection. Lobar pneumonia and pleurisy may less commonly occur. There may be protracted and severe conjunctivitis. Croupous laryngitis may occur. There may be otitis media.

In weakly children there occurs rarely gangrene of the cheeks or of the vulva (cancrum oris or noma). In some cases there is exhausting diarrhœa, which may assume a dysenteric character with bloody, slimy passages. True nephritis is rare, although albuminuria is common in the height of the disease. The disease may be complicated by whooping-cough.

Prognosis.—The prognosis is generally good except when severe epidemics occur in tenements, armies, and among savage races in virgin soil. Death seldom occurs from the disease itself, but from pulmonary complications. The prognosis is not good in children under the age of two years. The possibility of subsequent tuberculosis must be considered.

Treatment.—The patient should be isolated until desquamation is completed. Especial care should be exercised to to prevent delicate children with weak lungs or a tubercular predisposition from being exposed to the disease. The room should be of an even temperature (about 70° F.) and well ventilated. There is no advantage in keeping the room too hot. The patient should remain in bed until three or four days after the fever has gone, and during the febrile period should be kept on a milk diet. Water may be given freely. The majority of uncomplicated cases need no further treatment, though special symptoms may be treated as they arise.

The fever rarely needs treatment. Should it be high (over 104° F.), it may be reduced by sponging with water. Cool baths may be employed with benefit.

Conjunctivitis is best treated by careful cleansing of the eyes with a saturated boric-acid solution; or a few drops of a solution of atropia (gr. j : ℥j) or of alumnol (gr. v : ℥j) may be employed. For the redness of the eyelids the unguentum hydrargyri oxidi flavi (U. S. P.) may be used. In severe cases the room may be darkened slightly by blinds or by screens to relieve the photophobia.

Restlessness, delirium, and sleeplessness are best controlled by sodium bromide or phenacetine.

The cough, if troublesome, is best treated by paregoric and syrup of ipecac in small doses.

The itching of the skin may be relieved by washing with a solution of bicarbonate of soda or by oiling the skin with lard or with cacao butter.

Other symptoms as they arise are to be treated on general principles. During desquamation the skin should be oiled daily to prevent dissemination of the branny scales.

During convalescence great care should be taken to build the child up and to avoid most especially the least possibility of tubercular infection. Too much care cannot be taken in this regard.

RUBELLA.

Etiology and Synonyms.—Rubella is rather rare, occurring chiefly as epidemics, which are frequently extensive. Sporadic and endemic cases are exceedingly infrequent. It is a disease entirely distinct from measles, although closely resembling it in many of its clinical features. It is contagious to both adults and children, and one attack procures future immunity. *Synonyms:* German measles; Roseola; Rötheln.

Symptoms.—*Incubation.*—The period of incubation is usually two weeks.

Invasion.—The symptoms of the invasion resemble those of measles, but are much milder and are of shorter duration. There is a slight fever, rarely over 100° F., with headache, nausea and vomiting, coryza, sore throat, and swelling of the glands at the back of the neck that is almost characteristic. These symptoms rarely continue longer than twenty-four hours. In many cases they are so slight as to be unnoticed.

The eruption, which appears in from twenty-four to forty-eight hours, is first seen on the face and chest, thence spreading generally. It consists of small round, raised spots, of a pinkish rose-color, which are usually discrete and which frequently are seen on the palate. They are rarely crescentic. They may become confluent, the consequent reddening of the skin closely resembling the scarlatina rash; but the eruption is more erythematous, is not punctiform, and in places shows a papular character. In a certain number of cases there are developed from the papules a few vesicles which may become pustules. This is never the case with scarlatina nor with measles.

The eruption lasts for two or three days and then fades. There may be a slight branny desquamation.

During the eruption there may be some feverishness, an aggravation of the pharyngitis, and swelling of the glands at the back of the neck. In many cases, however, the only symptom is the eruption.

The **prognosis** is perfectly good.

Treatment.—Few diseases need so little treatment as rubella. The case should be isolated to avoid the spread of the disease.

EPIDEMIC PAROTITIS.

Definition and Synonym.—Epidemic parotitis is an acute contagious disease characterized by inflammation of the salivary glands. *Synonym:* Mumps.

Etiology.—Parotitis occurs both as an endemic and an epidemic disease, epidemics being usually extensive. It is a disease of childhood and adolescence, attacking infants and elderly people but rarely. It is more frequent among males than among females. It is personally contagious from the last few days of the period of incubation until the subsidence of the symptoms. The exact poison has not been absolutely proven, although a *bacillus parotidis* has been described.

Lesion.—The lesion consists in the swelling and congestion of one or of both parotid glands, and occasionally of the submaxillary glands as well.

Symptoms.—The period of incubation is between two and three weeks and is unattended by symptoms. The disease begins with fever—usually not over 101° F., but it may be as high as 103° or 104° F.—and attendant febrile symptoms, nausea, restlessness, and prostration. The local symptoms become noticeable in from twenty-four to thirty-six hours, although in some cases they may precede the fever. The patient complains of a feeling of tension, more rarely of actual pain with tenderness, referred to the parotid gland of one side. The gland is swollen, giving the patient a characteristically comical appearance. Deglutition and speaking aggravate the pain. There may be pharyngitis or earache.

The inflammation reaches its height in from three to six days and then subsides. It is usual for the inflammation to start on one side, the other parotid gland becoming affected in a day or so. More rarely both glands may be affected simultaneously, or the inflammation may be subsiding on the side first affected before the involvement of the other side, so that the duration of the disease is doubled. The lesion is rarely unilateral throughout.

In some cases the submaxillary glands may secondarily be involved. With the subsidence of the inflammation the fever and the constitutional symptoms disappear.

While the course is generally mild, there are cases which run a severe course, with high fever, rapid and feeble heart-action, and delirium.

Orchitis occurs more frequently in some epidemics than in others. It is rarely seen before puberty, and double orchitis is infrequent. The orchitis gives rise to pain and tenderness; the testicle is hard and swollen; there may be fluid in the tunica vaginalis.

The temperature rises, frequently to 103° or 104° F., and remains high until the orchitis begins to subside, which is usually in from five to ten days. Subsequent involvement of the other testicle may prolong the disease for another week. In females there may be vulvo-vaginitis, or the breasts may be enlarged and tender. Inflammation of the ovaries is rare.

Complications and Sequelæ.—There may be meningitis, mania, or post-febrile insanity. There may be suppuration or gangrene of the parotid glands. Severe orchitis may be followed by atrophy. In some cases arthritis has been observed. There may be deafness, which frequently is permanent.

Treatment.—The patient should be isolated until the disappearance of acute symptoms, and during the febrile period should be kept in bed and on a light diet. The parotiditis is best treated locally by applications of cold; should, however, heat be more grateful, poultices, applications of hot cloths, or dry cotton covered with oiled silk may be employed. Resolution may be hastened by applications of ichthyol ointment (ʒss : ℥j) or of iodine ointment.

Orchitis is to be treated by rest in bed, elevation of the testicle, and applications of cold. In the latter stages ichthyol ointment or unguentum plumbi iodidi may be employed with benefit. Other symptoms should be treated on general principles.

WHOOPING-COUGH.

Definition and Synonym.—Whooping-cough is an acute contagious disease with inflammation of the respiratory tract, a paroxysmal cough, and a characteristic "whoop." *Synonym:* Pertussis.

Etiology.—Whooping-cough occurs in endemic and epidemic cases, the epidemics being most frequent in the winter and early spring months, and often being associated with epidemics of measles. The disease is personally contagious, though concentrated and prolonged exposure is usually required. One attack procures immunity. The actual cause is probably due to a short bacillus growing in cultures with well-marked characteristics, first described in 1887 by Afanassjew. The majority of cases occur in children under six years of age, though no age is exempt. In negroes it runs a more severe course.

Lesion.—The lesion consists in a catarrhal inflammation of the nose, larynx, trachea, and bronchi. As complicating lesions there may be found extensive bronchitis of the smaller tubes, broncho-pneumonia, inflammation of the bronchial glands, and emphysema of the vesicular or interlobular variety.

Symptoms.—The *incubation* period of the disease is about two weeks, although this is difficult to determine owing to the insidious onset. The symptoms begin with bronchitis and coryza similar to those of an ordinary severe cold; these symptoms continue without improvement for from one to three weeks. Then appear the characteristic coughing attacks from which the disease is named. The attack begins with a series (fifteen to twenty) of coughs so rapid and spasmodic that the child cannot breathe. Suffocation seems imminent; the face is suffused; the eyes run; the tongue is cyanotic and protruding. The child is terrified and sits up in bed or runs to the nurse or the mother. After this series of coughs there is a long, deep inspiration with the sound of the characteristic whoop, immediately after which the convulsive coughs may be repeated. In some cases the whoop is the first indication of a coughing attack. In rare cases there is only the paroxysmal cough without any whoop. The attack is often followed by the raising of a little tenacious mucus, which gives relief.

Very severe attacks may be accompanied by vomiting or regurgitation of food, by relaxation of the sphincters, by convulsions, or by hemorrhages from the nose, mouth, stomach, or under the conjunctiva. In rare cases there may be symptoms of cerebral or subdural hemorrhage. The number of separate attacks varies from three to eighty during the twenty-four hours. The attacks are frequently induced by emotions, by crying, by attempts at swallowing, by close, dusty air, and by changes in temperature, and they are usually more frequent at night than during the day-time.

The general health suffers. Severe cases are accompanied by fever and prostration. The vomiting and the inducement of an attack by swallowing interfere with proper nutrition, while the child is nervous and fretful from loss of sleep. There is usually found superficial ulceration on each side of the frenum of the tongue, and between the attacks the face is frequently swollen, the lower lids are puffy, and the tongue is enlarged and of a bluish color.

After the paroxysmal stage has lasted for from three to six weeks the attacks become less severe, the whoop ceases, and there remains only a terminal bronchitis which slowly declines. The whole duration of the disease is in this way protracted for from six to twelve weeks. In adults the disease runs a more severe and energetic course, with marked depreciation in general health.

Complications and Sequelæ.—The pulmonary complications have been mentioned under the heading of *Lesions.* Their association with whooping-cough renders the prognosis much worse than if they occurred primarily.

Paralysis from cerebral or intradural hemorrhage is a rare sequela.

Whooping-cough is frequently followed by acute tuberculosis or by tubercular broncho-pneumonia, from an added infection of the inflamed lung or the bronchial glands. Quiescent tubercular deposits may also be called into activity.

In some cases there will be a return of the whoop at intervals for months whenever bronchitis is contracted. This is not a recurrence, but is merely a "habit-spasm."

Treatment.—Every possible care should be employed to keep children, especially if delicate, from being exposed. Negligence in this regard is criminal. The contagion is most marked during the paroxysmal stage, and declines with the terminal bronchitis, but does not persist after the second month. Isolation and quarantine cannot be insisted upon to their full limits, and a final disinfection of the rooms and the clothing is not necessary, as the contagion is conveyed by the breath alone. The room should be sunny and well ventilated. In mild cases without pulmonary complications the child can go out in favorable weather. The proper feeding of the child is important, especially if there be vomiting during the attacks.

Drug treatment is largely employed, and many alleged "specifics" are lauded from time to time. No one form of treatment is of service in all cases, but one drug after another has to be tried.

During the catarrhal stage the treatment is that of the bronchitis. During the paroxysmal stage treatment is directed not only toward the bronchitis, but also toward the mitigation of the paroxysms. For the latter purpose internal sedatives and local applications are employed.

Internal Sedatives.—The doses given are those suitable for a child two years old.

Belladonna, frequently to tolerance. Initial dose, 2 minims of tincture every three hours.

Quinine, in full doses, gr. j every two hours.

Chloral, gr. ij–iv every four hours : efficient in many cases, especially to secure sleep ; may advantageously be combined with sodium bromide (gr. iij–v), especially in children with convulsive tendencies.

Opium or *codcia* is to be used only in the severest cases.

Asafœtida mixture is often of service in mild cases.

Antipyrine (gr. i–iij every two or three hours) is often attended by most brilliant results.

Phenacetine (gr. ij–iij every three hours) has been found useful.

Bromoform (3 drops in simple elixir three times a day) acts almost as a specific in some cases.

The disinfection of the sleeping-room with sulphur, the child sleeping there in clean clothes after the room has been aired, often cuts short the paroxysmal stage.

Local treatment may be employed if not resisted by the child—inhalation of the vapor of carbolic acid, of creosote, atomized sprays of wine of ipecac, 1 per cent. solutions of resorcin, peroxide of hydrogen, or solutions of quinine.

Insufflations of quinine, 1 per cent. solutions of resorcin, or a powder of salicylic acid gr. x. to boric acid ℨij, may be tried.

In many cases, during the decline of the paroxysmal stage a change of climate may hasten recovery. In severe and protracted cases it should always be tried.

EPIDEMIC INFLUENZA.

Definition and Synonym.—Epidemic influenza is an acute infectious disease, occurring principally in pandemics, characterized by fever, pains, prostration, and by inflammation of the mucous membranes. *Synonym:* La Grippe.

Etiology.—The disease occurs in epidemics which spread over whole countries or quarters of the globe with great rapidity, attacking large numbers of people. The epidemics usually start in the East, the majority arising in Russia. Until 1870, ninety such epidemics had been described. The last pandemic occurred in 1889–90. The disease is not affected by sex, age, condition of life, climate, or atmospheric changes. Sporadic cases occur in the wake of the epidemics. One attack does not procure immunity. The disease is probably slightly contagious by personal contact, and possibly through fomites, but in the large epidemics infection cannot be traced with any certainty.

The actual cause of the disease appears to be a bacillus first described in 1892 by Pfeiffer. This is a short bacillus with bulbous ends that is found in the sputum, in the tissue of the lungs, and in the blood. This bacillus is never found in other diseases, is constant in grippe, and it causes the disease in monkeys and apes by inoculation.

Lesions.—The regular lesion is a catarrhal inflammation of the respiratory passages, from the nose to the bronchi; frequently there are also swelling and hyperæmia of the mucosa of the stomach and intestines.

Complicating lesions are numerous. The most important are those of the respiratory organs. There may be lobar pneumonia, broncho-pneumonia, pleurisy, or empyema.

Less frequently are noted abscess of the lung, abscess of the brain, meningitis, purulent pericarditis, and nephritis.

Symptoms.—The symptoms are exceedingly varied, depending on the severity of the infection, the reaction on the nervous centres, and the varying intensity of the catarrhal inflammations of the respiratory and gastro-intestinal tracts. Complicating lesions also add their symptoms.

1. *Symptoms of Infection.*—The onset is usually abrupt, being initiated by a chill or by chilly feelings. In some cases the chill is absent. Rarely there are prodromata, as evinced by lassitude of body and mind for several days. The temperature rapidly rises to 101° to 104° F. according to the severity of the attack. There is marked prostration from the first. The pulse is rapid and full. It may become feeble and intermittent, especially in the old and the debilitated, and may lead to heart failure. The liver and spleen in some cases are enlarged.

2. *Nervous Symptoms.*—At the onset there is severe, agonizing *headache*, usually frontal, less frequently general or occipital. This headache is often as severe as in meningitis, persisting for hours or days, and it may be obstinately present during convalescence.

Pain and tenderness of the muscles of the body, especially of the legs and the back, are constant symptoms. The patients feel bruised, and shift their position, trying to find one more endurable.

There are great *depression of spirits* and an inability to concentrate the mind which may last long into convalescence. In severe cases there may be delirium of an active type with hallucinations. There may be herpes labialis or urticaria.

3. *Catarrhal Symptoms.*—There are conjunctivitis, redness and œdema of the lids, and increased lacrymation. Coryza appears, with snuffling and sneezing. Laryngitis is attended by hoarseness, pain, and a severe paroxysmal cough which

often continues for weeks after the attack without being improved materially by medication. Pharyngitis and tonsillitis are usually present. The bronchitis may be mild or severe, and is marked by harassing cough, muco-purulent sputa, which may contain blood, pain in the chest, and the ordinary physical signs.

4. *Gastro-intestinal symptoms* consist in a heavily-coated tongue, persistent vomiting, diarrhœa, tympanites, and pain and tenderness over the abdomen.

Course of the Disease.—The relative predominance of the above symptoms varies in different people and in different epidemics. In all cases the symptoms of general infection are present, but the nervous, catarrhal, or gastro-intestinal symptoms vary so much in their relative intensity as to present three principal types of the disease—a nervous form, a catarrhal form, and a gastro-intestinal form. No strict line of demarcation between these different types can be made.

Complications and Sequelæ.—*Pneumonia* is by far the most frequent and fatal complication. There may be either broncho-pneumonia, especially in children, or lobar pneumonia. The latter, which involves one or more lobes, with complete or incomplete consolidation, and which is always associated with an intense general bronchitis, differs from the ordinary course of lobar pneumonia in the following particulars: The onset may not be so abrupt, being insidious and of slow development. The expectoration is that of bronchitis, rusty sputum being usually absent. The bronchitis is a marked feature, adding its symptoms and physical signs. The temperature is lower than that of a primary pneumonia, and is frequently remittent. Cyanosis and heart failure are more frequent. The duration is longer and resolution is more tardy. The temperature falls gradually, as a rule, and not by crisis, and frequently persists after resolution. The pneumonia is often "wandering," creeping from place to place until a considerable part of the lung has become involved. Empyema, pleurisy, abscess of the lung, meningitis, purulent pericarditis, and acute nephritis may occur during or after the attack, and give their regular symptoms.

Convalescence is usually slow and tedious, and is attended often by weakness of body and of mind. There may remain obstinate insomnia or cough. Facial neuralgia or headache may persist, and in some cases is due to complicating empyema of the antrum of Highmore. There may be post-grippal insanity or peripheral neuritis. Deafness may result from otitis media.

During and after epidemics of grippe the mortality from tuberculosis is manifestly increased, quiescent and chronic tubercular processes being stirred into activity.

The **duration** of an uncomplicated attack varies between three and seven days. It may be protracted by reason of the complications. Convalescence may be either rapid or slow and tedious.

Prognosis.—Grippe itself is rarely fatal except in elderly or debilitated people or in those suffering from advanced pulmonary, nephritic, or cardiac disease. In these cases the disease may terminate fatally from weakness or from cardiac failure. The prognosis is largely influenced by the nature and severity of the complications.

Treatment.—It certainly seems that the severity of the attack can be modified by the administration of large doses of quinine at the onset. At least gr. xx–xxx should be given within the first twenty-four hours, cinchonism being controlled by phenacetine or by sodium bromide. Aside from this the treatment is entirely symptomatic.

The headache and the pains in the bones and muscles are best relieved by repeated small doses of phenacetine, preferably combined with salol (each 5 grains every two hours). Antipyrine and antifebrine are to be avoided if possible, because of their depressing effect. Bromides may be useful in controlling restlessness, and a hot bath followed by a 10-grain dose of Dover's powder is often of great comfort. Morphine or codeine may be employed in severe cases. If headache depends upon congestion of the frontal sinuses, inhalations of steam, creosote, or menthol are of service. The following prescription is most satisfactory for the purpose:

℞.	Menthol,	gr. x;
	Tinct. benzoin. comp.,	℥j.—M.

Sig. A teaspoonful gradually added to a pitcherful of boiling water, to be inhaled three times a day.

Sleep is best procured by sulphonal, chloralamide, or codeia.

The vomiting is to be treated by regulating the feedings, by bismuth, or by morphine.

Diarrhœa is to be checked by opium combined with astringents.

The bronchitis is to be treated after the ordinary methods —by sedatives, by expectorants, and by counter-irritation applied to the chest.

Pneumonia requires its appropriate treatment, especial care being taken to avert heart failure by the timely administration of stimulants; alcohol, digitalis, and strychnine are most usually employed for this purpose.

The depressing and enfeebling influence of the disease during convalescence requires most careful attention. The patient should not return too soon to business; the diet should be supporting; all depressing influences should be avoided; and appropriate tonics should be administered. A change of air is often required. This supporting treatment is especially indicated in those who present evidences of tubercular disease.

DENGUE.

Definition and Synonyms.—Dengue is an infectious epidemic disease of warm latitudes, characterized by febrile paroxysms, pain in the muscles and bones, and anomalous eruptions. *Synonyms:* Dandy fever; Breakbone fever.

Etiology.—The disease appears in extensive epidemics confined to the tropics and the sub-tropics. It has occurred in the Southern United States. Large numbers of people are attacked, susceptibility being almost universal. One attack does not secure immunity. The disease is supposed to be contagious by personal contact and through fomites. Epidemics occur in the summer months, and are checked by colder weather. The exact poison has not been determined.

Pathology.—But little is known about the disease, as fatal cases are rare. There appear to be no essential lesions.

Symptoms.—The period of incubation is about four days. The onset is abrupt, beginning with a chill or chilly feelings, or with convulsions in children. The temperature rapidly rises to 102° to 106° F. according to the severity of the attack, and is accompanied by ordinary febrile symptoms. Cerebral symptoms are frequent in the cases with high temperature. At the onset are developed agonizing headache and backache. The muscles are sore and tender. The joints become painful, tender, and frequently red and swollen. The large and the small joints are equally affected. Prostration and depression are marked. In some cases there may appear a transitory erythematous rash. In rare cases there may be severe vomiting and purging. There may be hemorrhages from any of the mucous membranes in severe cases. Lymphatic enlargements are not uncommon. The febrile paroxysm lasts from three to five days and terminates by crisis, the fall of temperature frequently reaching the subnormal, although in most cases a moderate fever remains. At the crisis there may be sweating or diarrhœa. As the fever falls the general symptoms disappear, the patient feeling better, though often prostrated and sore.

The period of remission lasts from two to five days, and during it may appear a variety of eruptions which are not distinctive. There may be urticaria, erythematous eruptions of all kinds, or herpes. The severer forms may be followed by desquamation.

After the period of remission there occurs a second paroxysm of fever with a return of all the previous symptoms. This paroxysm, however, is mild and lasts only for two or three days, terminating again by crisis, after which convalescence is established.

Convalescence is usually slow and tedious from mental and physical incapacity.

The **prognosis** is almost uniformly favorable.

Treatment.—There being no specific treatment, the symptoms must be treated on general principles.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Definition and Synonym.—Epidemic cerebro-spinal meningitis is an acute infectious disease characterized by inflammation of the cerebral and spinal meninges. *Synonym:* Cerebro-spinal fever.

Etiology.—This disease, which has been recognized only since the early part of the present century, occurs chiefly in epidemics, although sporadic cases are frequently seen. The epidemics are most frequent and severe in the cold winter months, and are favored by poor hygiene and by the crowding together of people, as in garrisons and barracks. Children are more susceptible than adults. There is no evidence that the disease may be transmitted by food or by drinking-water. The disease is not considered contagious by either personal contact or through fomites, although rare cases have been reported which render it imprudent to make too dogmatic an assertion in this regard.

In almost all the recently-studied cases there is found in the exudate a lance-shaped coccus which appears identical with the diplococcus of pneumonia, and it appears most likely that this is the specific micro-organism of the disease. It is frequently found associated with the ordinary pus organisms.

Pathology.—The brain is usually congested. The veins and sinuses are engorged with blood. The pia mater is infiltrated with an exudate of fibrin, serum, and pus to a greater or lesser degree. The infiltration may be confined to the base or it may be more generally distributed. It is more abundant along the course of the blood-vessels and in the sulci. The lateral ventricles are filled with serum, which may be turbid from admixture of pus. In children, as a rule, and occasionally in adults, this fluid may distend and dilate the ventricles, and in chronic cases after the meningitis has subsided the distention of the ventricles may continue as a chronic hydrocephalus.

The pia mater covering the spinal cord shows similar inflammatory changes, especially on the posterior aspect. The brain-cortex is often infiltrated with pus, which may form small abscesses. The cerebro-spinal fluid, which is usually increased, may be turbid. In the exudate the lance-shaped cocci are found frequently with ordinary pus cocci. The lesions may involve the sheaths of the cranial nerves, leading to neuritis and perineuritis. In very malignant cases there may be no time for the lesion to develop before death.

In rare cases the meningitis is of the cellular variety. The pia may appear normal or lustreless or congested. There is neither fibrin, serum, nor pus, but there is a marked proliferation of the connective-tissue cells of the pia. These cases usually run a different clinical course.

The remaining lesions are not distinctive, being those common to all severe infectious diseases. There may be hemorrhages in the skin, in the serous membranes, and into the viscera. There is granular and fatty degeneration of the liver- and kidney-cells and of the heart-muscle. The spleen is usually enlarged and soft.

Symptoms.—The period of incubation is usually short, varying from a few hours to several days. During this time the patients may complain of headache, slight feverishness, and lassitude.

The onset is usually abrupt, being marked by a chill, fever, headache, and vomiting. The fever may reach to 102° or 104° F., and does not run any typical course. While high fever belongs to the severe cases, the reverse is not always true. In some cases the fever may not be marked.

The headache is usually frontal, but it may be parietal, occipital, or general. It is a severe headache, persisting during sleep and periods of stupor, as evinced by moaning, clasping the head with the hands, or by the facial aspects of pain. There may be general pains in the bones and muscles.

The vomiting is frequently severe and distressing, and does not depend upon the giving of food or of drink. It may assume a projectile character.

During the earlier stages of the disease there are nervous symptoms of irritation. The headache has already been mentioned. There are frequently psychical disturbances, as shown by delirium, which may be maniacal. Some patients show morbid erotic desires. There may be from time to time a sudden sharp cry, the so-called "hydrocephalic cry." The functions of the cranial nerves are exalted. There are photophobia, usually with some amount of conjunctivitis, intolerance to sounds, facial neuralgia, and muscular twitching.

The irritation of the spinal nerves is shown by pain, tenderness, and contraction of the muscles of the back of the neck that may amount to opisthotonos if the muscles of the trunk are similarly involved.

The skin is hyperæsthetic, the least touch causing exquisite pain. General exaggerated reflexes are highly characteristic. There are twitchings and spasms, and frequently automatic movements of the muscles of the arms or legs. The attitude is one of flexion.

The pulse is at first increased in proportion to the fever, becoming slowed and full when the brain begins to be compressed by the effusion and distention of the ventricles. It is often remarkably variable in its rapidity.

Various atypical eruptions may be seen on the skin. Herpes on the lips or the face occurs in half the cases. As the herpetic vesicles frequently contain the characteristic micrococci of the disease, bacterial examinations may be serviceable in confirming the diagnosis. There may be erythematous blotches or urticaria or petechial spots, which may be so grouped as to suggest a nervous origin.

The urine usually contains small amounts of albumin and casts. There may be polyuria. Glycosuria has been observed in a certain number of cases.

Digestive symptoms are not pronounced,.with the exception of the initial vomiting. The bowels are usually obstinately constipated. In a few cases a complicating dysentery has been observed. The abdominal wall may be markedly retracted, presenting a "boat-shaped" appearance. There may be severe abdominal pain.

In the latter stages of the disease the symptoms of nervous excitation give way to those of nervous depression, and symptoms of cerebral compression make their appearance. The patient usually makes no more actual complaint of headache, although it is evident that there is still some pain experienced. There are increasing dulness and apathy, which may proceed to stupor and coma. There may be periods of delirium, usually now of the low, muttering variety. Photophobia is succeeded by lack of perception of light; the pupils are usually dilated and do not react. Noises are not objectionable. The muscular twitchings and spasms give way to weakness and paralysis, which are most marked in the face and in the eye-muscles, producing strabismus. The temperature continues to be irregular. The respiration may be irregular, or even of the "Cheyne-Stokes" variety. The pulse is usually slower than would be expected from the prostration and the fever, becoming rapid and feeble, however, toward the close of the disease. The combination of fever, headache, slow pulse, and constipation is exceedingly characteristic of meningitis in any of its forms. At the close of the disease there may be diarrhœa and loss of the sphincter control. In fatal cases there may be ante-mortem hyperpyrexia. In cases that recover the fever gradually disappears, while other symptoms depart more slowly.

The duration of ordinary cases is between one and three weeks, although more protracted cases are not uncommon.

Anomalous cases are met with, especially in certain epidemics.

1. *Course in Young Babies.*—Convulsions are frequent at the outset and throughout the disease; the fever is high; the pulse is rapid throughout; the child passes into stupor alternating with restlessness, and death in coma ensues.

2. *Mild Cases.*—There are headache, nausea, vertigo, and

a little fever. There may be stiffness of the neck and vomiting. The diagnosis of these cases is difficult except during epidemics.

3. *Intermittent Cases.*—The regular course of the disease in these cases is marked by periods of improvement and remission of fever lasting for a few hours or days, the remissions recurring at regular intervals. The case may be mistaken for malaria or pyæmia.

4. *Abortive Cases.*—Here the disease begins in the regular way, but recovery is rapid after a few days.

5. *Malignant Cases.*—The patient is suddenly attacked by a chill, headache, and high fever. The pulse is feeble and frequently is slow, and hemorrhagic spots usually appear on the skin. Cerebral symptoms develop rapidly, and death from toxæmia occurs in a few hours or days, before lesions or characteristic symptoms have time to develop. These cases are usually seen only in the most severe epidemics.

Complications.—There may be bronchitis or bronchopneumonia. Lobar pneumonia is frequently observed. There may be pericarditis, endocarditis, or pleurisy. There is usually conjunctivitis, but more serious lesions may develop, such as purulent keratitis or choroiditis with loss of sight, or optic neuritis with atrophy. Arthritis occurs in some epidemics, the joints being painful, red, and swollen from effusion in and around the joint-cavities, the effusion in some cases being purulent.

Sequelæ.—Convalescence, which is apt to be slow and tedious from prolonged muscular and mental weakness, may be complicated by relapses. In some cases the lesion in the lateral ventricles continues as a chronic hydrocephalus. The patient recovers partially from the acute attack, but a little fever remains, the pulse is feeble, and there is great gastric irritability. The pupils are usually dilated. Emaciation becomes extreme. The patient lies in apathy or stupor varied by periods of restlessness and moaning. These cases last for weeks, and final recovery is rare.

Some patients are left in a condition of bodily and mental weakness. They become anæmic, irritable, forgetful, and hysterical, usually recovering after a lapse of months.

There may be paralysis from post-febrile neuritis.

There may be sequelæ from involvement of special senses. There may be partial or complete blindness from corneal ulcerations, from choroiditis, or from atrophy of the optic nerve. Permanent deafness may result from perineuritis of the auditory nerve or from labyrinthine disease, many cases of deaf-mutism being due to this disease.

The **prognosis**, which is bad, but not hopeless, depends not only on the severity of the symptoms, especially those of cerebral origin, but also on the general character of the epidemic, the mortality ranging from 20 to 75 per cent. in different epidemics. The average mortality is about 40 per cent. Endemic cases are usually less severe than those occurring during epidemics.

Treatment.—The treatment is entirely symptomatic. In robust patients, if seen early at the outset, local bleeding by leeches applied to the temples or behind the ears, or the application of wet cups to the nape of the neck, is frequently of much benefit. Relief is usually afforded by the continuous application of cold, by the cold coil, or by ice-bags applied to the head or the spine. Large doses of ergot are frequently given in the earlier stages to diminish cerebral congestion, and iodide of potassium in 20-grain doses three times a day is warmly recommended throughout for its supposed "absorbent" action.

Blistering is now considered injudicious, as it does no good and adds to the discomfort of the patient. Applications to the shaven scalp of iodoform ointment and mercurial inunctions have not seemed to exert a favorable effect.

For the nervous symptoms sedatives and even narcotics are required. Bromide of sodium, hyoscyamine, phenacetine, or small doses of atropine may be enough in mild cases, but the severer forms demand morphine, preferably by hypodermatic administration.

High temperatures are to be combated by hydrotherapy, and every indication of heart weakness must be met by the free and judicious use of heart stimulants.

DIPHTHERIA.

Definition.—Diphtheria is an acute contagious disease due to infection by the Klebs-Loeffler bacillus, and characterized by an exudative inflammation of mucous membranes or of abraded cutaneous surfaces, with constitutional symptoms.

Etiology.—The disease is endemic in all large cities, and frequently becomes epidemic, the most characteristic epidemics occurring in summer hotels, in institutions, and in small villages. The spread of the disease is greatly favored by poor hygiene, bad plumbing, and the crowding together of people. It is contagious by personal contact, and may be transmitted by clothing, toys, bedding, etc., even after the lapse of months or of years, the germ having intense vitality and duration of life. Cases, however, in which the contagious property of the bacillus has been retained for more than one year, though reported in medical literature, are of questionable authenticity.

It is doubtful whether the disease is communicable by the breath alone. It is known, however, that the greatest contagion is conveyed by the secretions and by loosened bits of membrane from the infected site coughed into the faces of the attendants or sucked from the tracheotomy wound by an over-zealous operator. The disease may be transmitted by kissing. Cases are reported in which the disease has been taken from diphtheritic animals, especially cats.

The disease is usually one of childhood, one-half the cases occurring before the fifth year, although it is common enough in those under fifty. It may occur in young babies. One attack does not procure immunity. The exciting cause is now known to be a bacillus described in 1883 by Klebs and in 1884 by Loeffler, and bearing their combined names. Its length is a trifle less than the tubercle bacillus, but it is broader, with clubbed extremities. It is readily stained, shows a characteristic growth in nutritive media, and is capable of causing the disease in animals. Cultures are best made in blood-serum, colonies in agar-agar. It is destroyed by aqueous solutions of bichloride of mercury (1 : 8000), salicylic acid (1 : 2000), and carbolic acid (1 : 50). It is destroyed also by boiling. The bacilli are usually found only in the pseudo-membrane, though exceptionally they may be present in the blood and in the viscera. They may persist in the throat for so long as three weeks after the attack, and are found in the throats of 25 per cent. of those who have been exposed to diphtheria. They are never found in other diseases.

Infection usually occurs through slight abrasions of the mucous membranes, and is favored by diseased conditions of the upper air-passages. The diphtheritic ptomaïne has been isolated, and its injection in animals has been followed by all the symptoms of diphtheria except the membrane.

Immunity.—By the inoculation of attenuated cultures in some animals immunity has been secured; and, what is more important, infected guinea-pigs have been cured by inoculating them with the blood of the animals rendered thus immune. At the present time diphtheria in the human subject is being treated by this method, with the result of reducing the mortality one-half. If these inoculations are begun by the second day of the disease, the patient almost invariably recovers.

Pathology.—The *essential lesion* consists of a croupous inflammation of mucous membranes, more rarely of abraded cutaneous surfaces. The most frequent sites are the tonsils, pharynx, palate, nares, larynx, trachea, or bronchi, and less frequently the mouth, gums, lips, œsophagus, stomach, and vagina.

The mucous membrane is congested, swollen, and infiltrated with fibrin-serum and pus, which appear on its free surface. The epithelial cells and the exuded leucocytes die and undergo hyaline degeneration, losing their nuclei—the so-called "coagulation-necrosis" of Weigert. There may be necrosis of the false membrane, and of the stroma of the underlying mucous membrane as well, which may become gangrenous in some cases. Erosion of large arteries with even fatal hemorrhage may result.

If the patient recovers, the false membrane sloughs off *en masse* or by gradual disintegration, superficial ulcers being usually left. If the ulcers be deep, evident cicatrization may result. Successive crops of membrane form if the false membrane be forcibly detached.

In the false membrane are found the characteristic bacilli associated with a variety of other organisms, especially streptococci and staphylococci, which, as a rule, penetrate more deeply than does the Klebs-Loeffler bacillus.

The appearance of the membrane varies. It may be adherent, a bleeding surface being left after its forcible removal, or it may be shreddy and readily detached. It may be thick, soft, and yellow, or it may be thin and so transparent that it can hardly be seen by the naked eye, and in some cases there may be evident only a localized hyperæmia. It may be of a dirty-green color, or it may be putrid and gangrenous. The surrounding mucous membrane is congested and inflamed. These appearances are identical with those of pseudo-diphtheritic membranes, the only point of difference being the presence of the Klebs-Loeffler bacilli in the true cases.

Complicating lesions are variable. There may be adenitis of the lymphatic glands near the infected area, which may proceed to suppuration from mixed streptococcus infection. The periglandular tissues, and even the salivary glands, may become in like manner affected. Bronchitis, either catarrhal or diphtheritic, areas of atelectasis, and patches of bronchopneumonia are usually present in fatal cases. There may be endocarditis in rare cases, but an acute degeneration of the heart-muscle is not uncommon and may lead to sudden death.

The kidneys may be the seat of an acute degeneration or of an exudative or a diffuse nephritis. The spleen may be found large and soft. In cases fatal from asphyxia the viscera are usually congested.

Incubation may occupy from one to fourteen days, the average duration being from two to five days.

The **symptoms** may be divided into two groups: 1. General symptoms due to the ptomaïne-poisoning, and which are the same in all cases; 2. *Local symptoms*, which vary according to the localization of the lesion.

1. *General Symptoms.*—In some cases the disease begins abruptly with a chill or, in children, with convulsions. Usually, however, the onset is insidious, being marked by prostration, fever, and often by digestive disturbances.

The fever does not run a typical course; it may be as high as 104° F., but a temperature of from 101° to 103° F. is more common. It is often irregular or intermittent, and possibly is altogether absent even in fatal cases.

Prostration is an early and constant symptom, and is proportioned more to the actual gravity of the case than to the height of the fever or to the local lesion. In mild cases prostration may be slight or absent. The pulse is rapid, with a tendency to become feeble according to the severity of the disease. In some cases the pulse may be slow (50 to 60); this is not usually a favorable sign. At any time sudden or gradual heart failure may develop, even during advanced convalescence. This makes the prognosis uncertain in every case.

Sudden heart failure will be followed by almost instantaneous death. Gradual heart failure will be shown by increasing rapidity and weakness of the pulse, dyspnœa, cyanosis, and congestion of the different viscera, with death in a few hours or days.

Cerebral symptoms comprise stupor, often alternating with restlessness, or mild delirium or convulsions, semi-coma, and coma. These symptoms are rather rare considering the severely toxic character of the disease. They may appear early, from the toxic action of the ptomaïnes on the nervous centres, or they may appear late, as the result of asphyxia.

In some cases there appears an erythematous eruption resembling that of scarlatina. It is, however, evanescent, fading usually in a few hours. Bacterial examination of the pseudo-membrane or the exudate may be necessary to differentiate between this disease and those cases of scarlatina complicated with pseudo-diphtheritic pharyngitis. In malignant cases there may be purpura. Albuminuria occurs in the majority of severe cases, from parenchymatous degeneration of the kidney. The occurrence of a true nephritis must be considered as a complication.

2. *Local Symptoms.*—(*a*) *Tonsillar Diphtheria.*—This is the commonest form of diphtheria, and at the same time the least serious. There are three clinical forms:

(1) There is a pseudo-membrane on one or both tonsils, having no relation to the crypts. (2) The crypts of the

tonsils are filled with a pseudo-membranous exudate which appears on the surface as white points, resembling in every way the appearances presented by ordinary follicular tonsillitis, and from which it can be differentiated only by bacterial examination. These white points in some cases may so coalesce that the tonsils are covered with irregular white patches. (3) The tonsil swells; there are swelling and œdema of the surrounding structures, resembling the appearances of ordinary suppurative peritonsillitis or quinsy. No membrane is visible until after thirty-six to forty-eight hours. These cases, which seem to be due to bacterial invasion of the deeper structures, are apt to do badly.

The *local symptoms* of the first two varieties are apt to be mild, lasting but a few days. There may be moderate fever; prostration is slight or absent; the voice is muffled; there are pain and soreness, which are increased by talking or swallowing.

However mild the case, the disease may spread and become severe, or it may be followed by any of the complications or sequelæ, and it may be the cause of infecting others even with the most severe forms.

(*b*) *Pharyngeal Diphtheria.*—(1) There may only be an area of local hyperæmia without any pseudo-membrane. This condition is seen in those exposed to diphtheria, and is commonly called "sympathetic sore throat." It is really diphtheritic, however, and may not only be followed by sequelæ or the spread of the disease, but may even be the source of contagion to others.

(2) There may be a pseudo-membrane evident, usually associated with membrane on the tonsils.

Symptoms.—In mild cases there may only be malaise, slight fever, and a raw feeling in the throat. In severer cases there may be pain, increased by talking or by swallowing, muffled voice, fetid breath, and in some cases ptyalism.

(3) *Nasal diphtheria* is not common. It is rare as a primary form, being usually secondary to membrane in the pharynx. From the formation of pseudo-membrane in the nasal cavities and the attendant swelling of their mucous membranes the nostrils become more or less occluded. There is usually a discharge from the nose of muco-pus or sero-pus, which may be stained with blood. In some cases there is a brown, watery discharge which stains the pillow and excoriates the lips.

The glands of the neck are more often involved in naso-pharyngeal diphtheria than in any other form; considerable deformity usually resulting from their tumefaction.

If the nose be involved, the patient is apt to do badly. Death usually results from sepsis with cerebral symptoms or from heart failure. Small babies either asphyxiate or starve from their inability to breathe if they are nursed, unless they are fed by the stomach-tube.

(4) *Laryngeal diphtheria* is a common form, and is greatly dreaded for the following reasons:

(*a*) There is apt to be asphyxia from the occlusion of the glottis by pseudo-membranes, by the swelling and œdema of the vocal cords, and in some cases by their paralysis, from the projection of little tongues or tags of loose pseudo-membrane into the rima glottidis, and by the spasm from time to time of the laryngeal muscles.

(*b*) As the larynx is seldom infected primarily, but is affected from the spread of the disease from the pharynx, the pseudo-membrane is apt to be extensive and toxæmia severe.

(*c*) The pseudo-membrane is apt to spread downward and to involve the trachea and the bronchi.

(*d*) There is apt to be developed either septic broncho-pneumonia or "deglutition-pneumonia" or areas of atelectasis.

(*e*) Because the larynx is affected usually in children, who do not stand the disease well.

Symptoms.—The voice is hoarse and croupy, and may be reduced to a faint whisper. There is a hoarse, croupy cough. The breathing is rapid and inefficient, and there is obstructive dyspnœa which may be either obvious or masked.

In obvious dyspnœa the child sits up with the neck craned forward, to bring into play all the accessory muscles of respiration. The alæ nasi dilate; inspiration is prolonged and stridulous. There is inspiratory sinking of the spaces above and below the clavicles. The face is anxious and distressed, and may be semi-cyanotic. From time to time there occur paroxysms of increased dyspnœa that are often relieved by coughing up pieces of pseudo-membrane. Unless the condition is relieved by the casting off of the pseudo-membrane or by operative interference, the child passes into the condition of masked dyspnœa.

In masked dyspnœa the child no longer struggles for breath, but lies flaccid or in a stupor which may deepen into coma. The skin becomes cold and livid, the pulse becomes more rapid and feeble. In this condition the patient may remain for from one to four days, and recovery from this stage is exceedingly rare. All cases, however, do not pass into this stage, but after a time the pseudo-membrane becomes loosened and is coughed up either in large pieces or by gradual disintegration. Glandular swellings are not seen in diphtheria of the larynx alone.

Complications and Sequelæ.—Local complications have already been alluded to. They are sloughing, erosion of arteries with hemorrhage, and swelling of the neighboring glands.

Pulmonary complications occur in almost all fatal cases, and present their ordinary symptoms and physical signs.

Should pneumonia occur in very sick patients, its symptoms are frequently masked by those of the primary disease.

There may be gastritis or enteritis. Renal complications are common.

Acute parenchymatous degeneration shows itself by changes in the urine alone. Acute exudative or acute diffuse nephritis may occur during the disease or during convalescence. There are the changes in the urine common to such lesions; there may be suppression with uræmic symptoms. In some patients much exhausted by the disease uræmic symptoms may not appear.

Of the sequelæ, peripheral neuritis is the most important. It is seen in from 10 to 40 per cent. of all cases, according to the epidemic, and usually occurs in the second or third week of convalescence. It may follow either mild or severe cases.

The muscles most frequently paralyzed are those of the soft palate. The voice becomes nasal, there is inability to clear the throat, and deglutition is interfered with, fluids regurgitating through the nose. This may be the only symptom. The patient may have to be fed through the stomach-tube, especially if the paralysis extend to the constrictors of the pharynx.

The next most common forms are paralyses of the eye-muscles. The intrinsic muscles may be affected, causing dilatation of the pupil and loss of power of accommodation. The involvement of the extrinsic muscles produces ptosis and strabismus.

If the muscles of the larynx be affected, there will result dyspnœa, aphonia, and a croupy cough.

More rarely the muscles of one or more extremities may become paralyzed, or there may only be some weakness in the legs with loss of tendon-reflex.

Diphtheritic paralysis usually lasts but a few weeks or months, terminating in recovery, although in some cases it may become permanent.

The nephritis may become chronic and lead to the death of the patient. There may be resulting endocarditis. If the conjunctivæ be the seat of a pseudo-membrane, ulcers of the cornea with opacities usually result.

The **prognosis** is always serious, and must in all cases be made guardedly, because, however mild the attack may be, it may at any time spread and become severe. The danger of heart failure must always be regarded. The prognosis depends upon the character of the prevailing epidemic and also upon the age of the child, 42 per cent. of cases being fatal before the fourth year, 35 per cent. between the fourth and tenth years, and 10 per cent. between the tenth and twentieth years. The average mortality is about 25 per cent. The prognosis depends not only upon the severity of the general infection, but also upon the locality and extent of the lesions, being worse in nasal, laryngeal, pharyngeal, and tonsillar cases in the order mentioned. The prognosis also depends upon the presence of complications and upon possible sequelæ. As a rule, a good prognosis may be given to cases of tonsillar diphtheria that do not spread within two days.

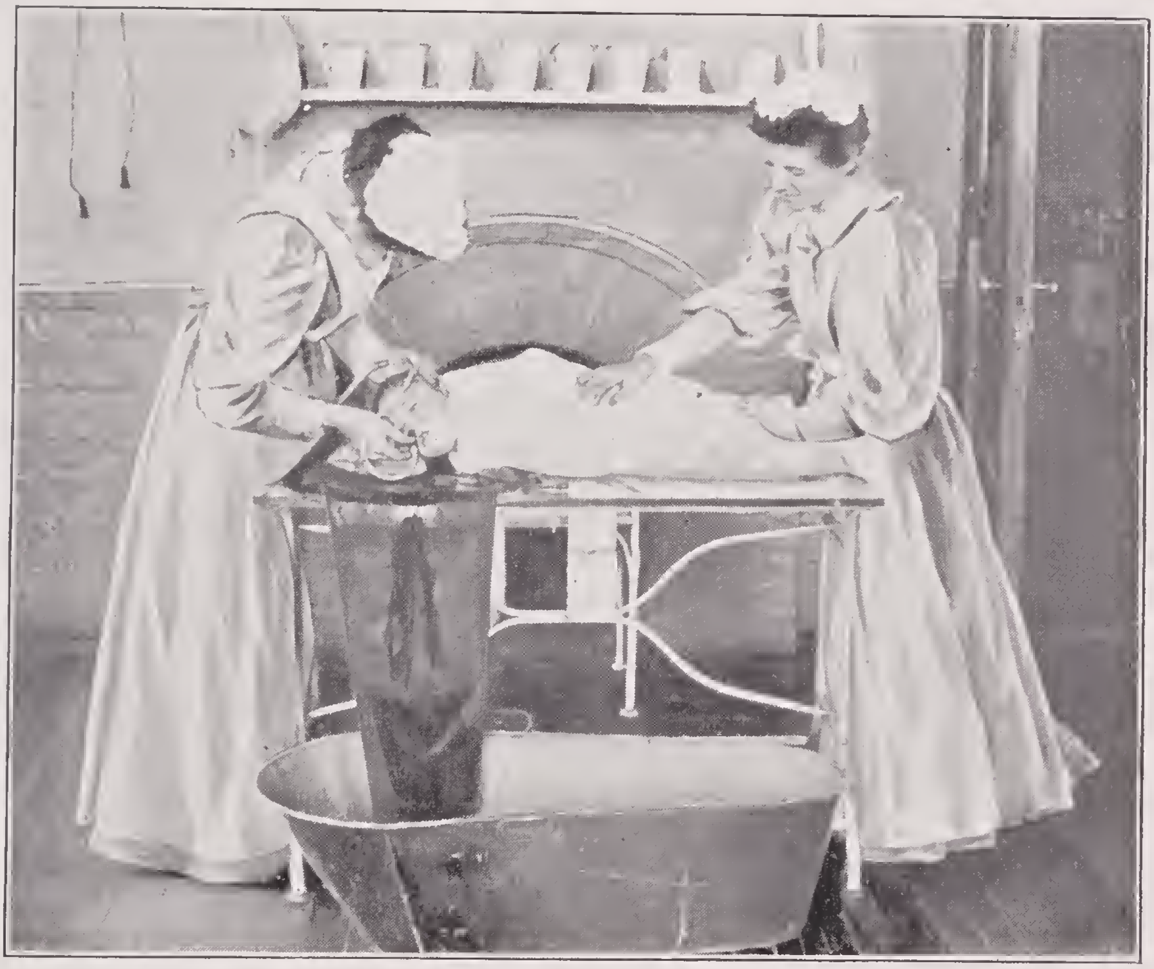
The mortality has been reduced within the past year, under the antitoxine treatment, to just one-half the former death-rate. If cases can be treated by antitoxine within the first thirty-six hours, the mortality will still further be reduced.

Treatment.—*Prophylactic Treatment.*—Careful isolation should be enforced, not only for the patient, but also, as far as possible, for the attendants. "The members of a household in which a case of diphtheria exists should be regarded as sources of danger unless cultures from their throats show the absence of virulent diphtheria bacteria."

(Park). Attendants on the sick should have their throats irrigated every four hours with a 1 : 10,000 bichloride solution. Isolation should be continued until the bacilli are proved to be absent. In one-half the cases the bacilli disappear within three days after the disappearance of the pseudo-membrane; in one-third of the cases, in seven days; in one-tenth of the cases, in fourteen days; while the bacilli may remain in a small percentage of cases as late as the sixty-third day. After isolation is relaxed, the room, with the bedding, toys, etc., should be fumigated thoroughly with sulphur, and the linen should be boiled in 2 per cent. carbolic solution. The walls and the floors should be scrubbed with 1 : 10,000 bichloride solution.

General Treatment.—The child should be put to bed and be kept on a milk diet. The temperature of the room should be 75° F., and the air should be kept moist. The use of the steam-tent will be alluded to. Careful attention should be paid to the heart, and stimulants should be given freely when required. Especially are alcoholic stimulants to be recommended. The danger of heart failure during early convalescence should not be forgotten. The temperature may be controlled, if necessary, by hydrotherapy. Internal antipyretics should be avoided. Large repeated doses of the tincture of the chloride of iron (4 to 5 drops hourly to a child of three years) and the use of corrosive sublimate in small doses (gr. $\frac{1}{16}$ every four hours as a limit, or gr. $\frac{1}{60}$ every hour) have been recommended warmly. The former is recommended, with alcoholic stimulants, as a routine treatment. Chlorate of potash should not be employed, because of its evil effect upon the kidneys.

Local Treatment.—As diphtheria is a local disease at first, the bacilli growing in the pseudo-membrane and elaborating there the toxalbumins that poison the system, local treatment becomes of the first importance in destroying the activ-



Irrigation of naso-pharynx (Berg).

ity of the germ and in removing the toxalbumins when formed. Rough treatment and the mechanical tearing off of the pseudo-membrane are of actual harm. The best treatment consists in the thorough irrigation of the throat and nose with a neutral salt-solution (ʒj to Oj) or with boric-acid solution, a tablespoonful to the pint. A fountain syringe should be used, and the fluid should be at a temperature of 85° F. The patient lying on the side (as in Plate 9), the nozzle of the irrigator should be inserted in the upper nostril until the fluid runs out of the other nostril and the mouth. Then the process is repeated with the lower nostril. The tip of the irrigator should then be passed well back over the dorsum of the tongue, and the pharynx well flushed out. In case the nostrils be occluded with membrane, the fountain syringe may be elevated six or eight feet. Such irrigation should be done every two hours. In septic cases irrigation with bichloride (1 : 4000 to 1 : 8000) may be employed every four to eight hours in addition. If the membrane be thick, insufflations of papoid powder may be used to advantage. Other local remedies have been recommended, but are not now as much used as formerly. Among these remedies are insufflations of powdered sulphur, applications of tincture of the chloride of iron, iodine, peroxide of hydrogen, trypsin, and lactic acid.

So high an authority as Loeffler recommends the following method of local treatment: After cleansing the surface of the pseudo-membrane the following solution is to be applied on cotton swabs for ten seconds every three hours, later three times a day:

℞. Alcohol,	60 volumes;
Toluol,	36 volumes;
Liq. ferri sesquichlorati,	4 volumes;
Menthol,	20 volumes.

In laryngeal diphtheria with dyspnœa two additional remedies are warmly recommended—the steam-tent and calomel fumigation.

The steam-tent should be used upon the first appearance of dyspnœa, but it may be used in any case as a routine measure. A good way to form the tent is to throw sheets over clothes-horses arranged about the bed, or to suspend from the ceiling an open umbrella about which the sheets can be draped. The nozzle of a steam-kettle should be inserted within the tent, so that the air is kept constantly moist.

Calomel inhalations are employed in laryngeal diphtheria, especially with dyspnœa: 10 to 20 grains may be used every two to four hours without danger of salivation to a child, although the nurses may suffer from sore gums; mercuric diarrhœa may, however, occur. The calomel should be piled on a piece of tin resting upon the sides of a small pail or chamber utensil, under the centre of the tin being the alcohol lamp (Fig. 1). The whole apparatus is to be placed inside the croup-tent, and care must be exercised that the child does not kick the lamp over and set fire to the bedding.

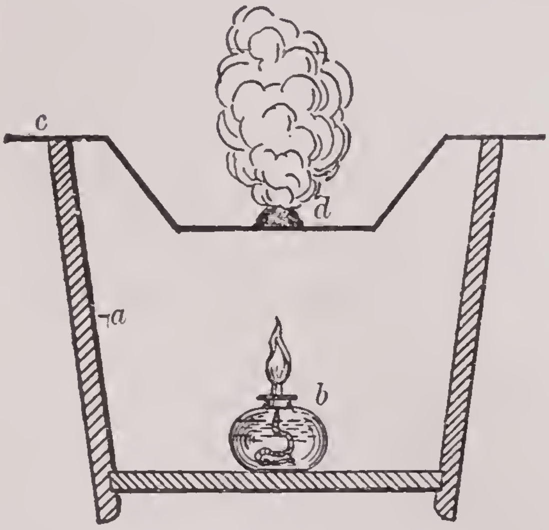
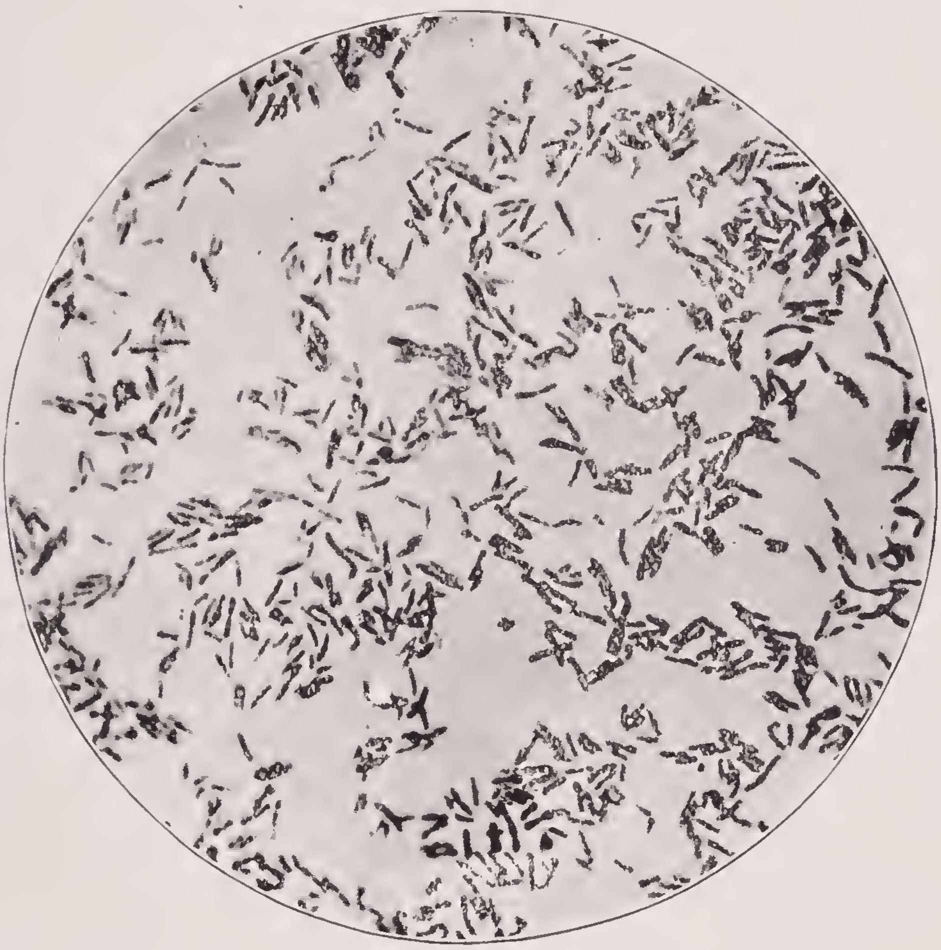


Fig. 1.—Method of fumigating with calomel: *a*, pail (section); *b*, alcohol; *c*, strip of bent tin; *d*, pile of calomel powder.

When the laryngeal pseudomembrane is being loosened, its separation may be hastened by emetics; but these are not to be recommended except in robust cases with good heart-action, in whom dyspnœa appears due to the obstruction caused by the loose membrane and the mucous secretion.

1



2

1. Klebs-Loeffler bacillus (photographed by Dr. W. H. Park). 2. Hutchinson teeth.

When actual obstructive dyspnœa begins the question of operative interference comes into consideration. For the details of intubation and tracheotomy the reader is referred to works on surgery. It seems that intubation should be our first choice, and it should be done as soon as slight cyanosis appears.

Serum-therapy.—The growth of the Klebs-Loeffler bacillus (Plate 10) in the body-tissues develops the peculiar toxalbumin to the poisoning from which the constitutional symptoms are due. Nature in some unknown way elaborates in the body an antidotal poison, the antitoxine, and when the two poisons balance in effect the constitutional symptoms cease and the patient recovers. If antitoxine can be made outside the body, and injected when needed, in sufficient doses, without waiting for the system to elaborate it, the disease, it was thought, might be stamped out at the onset. The growing experiences of the past year tend more and more to prove the correctness of this assumption.

The method by which the antitoxine is obtained is as follows: All animals susceptible to diphtheria can be rendered immune by injecting into their bodies pure cultures of the bacillus in gradually increasing virulence. Weakening of the toxine is effected by diluting it with iodine solutions. For various reasons the horse is the most satisfactory animal for the purpose.

When immunized, the blood-serum contains a large quantity of antitoxine and can be used for subcutaneous injections in human diphtheria. A small quantity of tricresol is added to the serum to keep it from decomposing. Three reliable serums are in the market—Roux's, Behring's, and Aronson's. Each serum supplies a weaker (" immunization serum ") and a stronger solution.

The dose of antitoxic serum varies with the size and the weight of the patient, the virulence of the disease, and the length of time since the onset of the symptoms. The ordinary dose of the weaker or immunization serum is 20 cubic centimeters as a single injection, given in the skin between the shoulders or in the flank. A subsequent injection of 10 cubic centimeters may be given in twenty-four hours if the Klebs-Loeffler bacilli are found present. Ordinarily two such injections suffice for recovery. A red rash may follow such injections. Under this treatment the temperature rapidly falls, the membrane rapidly disappears, and early convalescence is established (see Fig. 2).

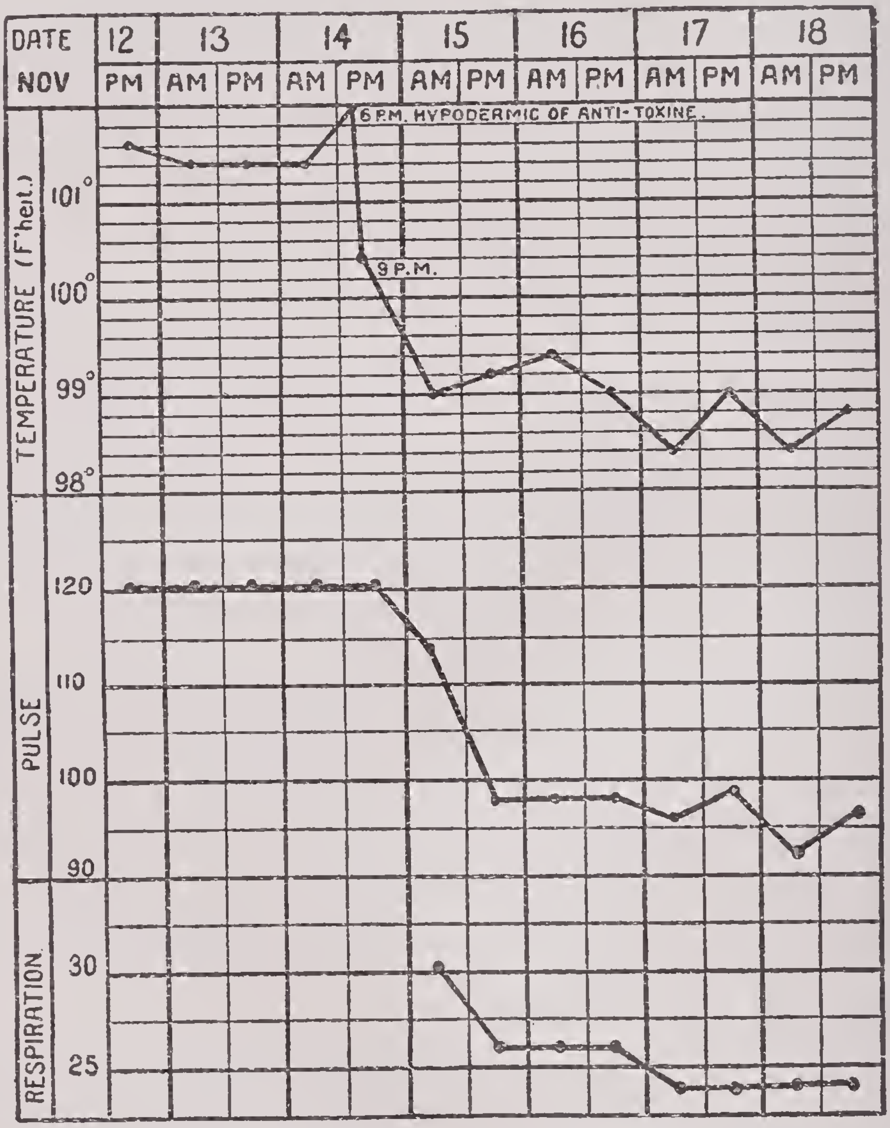


Fig. 2.—Chart of diphtheria treated by antitoxine (Fischer).

It is believed that recovery should take place in all patients treated within the first twenty-four hours. Taking all the cases together, the mortality has been reduced one-half, and with improved methods and improved appreciation of the value of early diagnosis and early treatment the mortality will still further be reduced.

ERYSIPELAS.

Definition.—Erysipelas is an infectious inflammation of the skin with constitutional symptoms, caused by the inoculation of the streptococcus erysipelatosus.

Etiology.—The disease is due to infection of the lymphatics of the skin by a streptococcus which is identical in appearance with the streptococcus pyogenes. Infection always occurs through wounds or abraded surfaces, intact skin and mucous membranes affording absolute protection. Two forms are usually described—(*a*) a *traumatic* form complicating surgical wounds and injuries, and (*b*) an *idiopathic* form occurring usually in the face, in which no traumatic point of origin can be found. The only real difference between these two forms is that in the one the point of entrance is apparent, while in the other it is slight and is usually overlooked. The disease is favored by poor hygiene, by bad plumbing, and by contact with infected cases. As inoculation through abraded surfaces is necessary to cause the disease, it cannot be considered as contagious in the ordinary acceptation of the term. It is commonest in alcoholic and debilitated patients and in those suffering from Bright's disease. One attack does not secure immunity. Some patients are exceedingly susceptible and may suffer from repeated attacks.

Pathology.—Erysipelas is really a progressive lymphangitis of the skin involving the perilymphatic tissues by continuity. There is an infiltration of the cutis vera by fibrin serum and leucocytes; this infiltration in severe cases may extend to the subcutaneous connective tissue. The lymphatics are crowded with the streptococci, especially at the margin of the patch and extending into the healthy skin. Vesicles and bullæ may be formed. Suppuration does not occur unless there be an added infection by pus microbes.

Symptoms begin from fifteen to sixteen hours after inoculation.

General Symptoms.—The disease usually is initiated by a chill and a rise of temperature to 103° or 104° F. or even higher. Nausea and vomiting are common at the onset. The pulse is full and bounding, and is rarely over 100 except in the most severe forms or in debilitated and alcoholic subjects. In these cases the pulse may become rapid and feeble and may be a source of real danger.

Delirium belongs to the severer cases, and is especially marked in alcoholic patients. It may be either mild and maniacal or low and muttering. There is usually severe headache, especially in erysipelas of the face and scalp. Prostration is marked in proportion to the severity of the disease. There may be albuminuria. In fatal cases the patient may pass into a "typhoid condition."

The constitutional symptoms may be mild, such as would be due to a slight local inflammation, or they may be exceedingly well developed, resembling those caused by any severe general infection.

Local Symptoms.—The skin becomes swollen and shiny and of a rose color which disappears on pressure. It is distinctly thickened and indurated. The patient complains of feelings of tension, burning, and itching. The swelling is most marked in places where there is considerable loose connective tissue, as in the face or the eyelids, and in these localities there is also considerable œdema. Inflammatory changes are more marked at the border of the eruption, which is abrupt and elevated and shows tongue-like prolongations projecting into the healthy skin. These characteristics of the border of the patch are distinctive and absolutely diagnostic. In some cases there appear vesicles or bullæ.

If there be an added infection by the ordinary pus microbes, the contents of the vesicles or the bullæ may become purulent, or there may be complicating phlegmonous inflammation of the deeper structures of the skin, or metastatic abscesses of internal viscera, or septic inflammation of any of the serous membranes. In some cases the local inflammation is so intense that superficial gangrene results. Exfoliation of the skin usually follows the subsidence of the eruption.

When the disease once begins, it is common for it to spread from the periphery, so that large surface areas may become successively involved. Its progress may be checked by any decided fold in the skin, particularly the naso-labial fold. It is frequently limited at the border of the hairy scalp. In facial erysipelas the chin and the anterior aspect of the neck are never affected. In some cases erysipelas shows a tendency to creep from place to place, subsiding in the old situations while new areas are constantly becoming invaded. In this way it may spread over most of the body and the extremities. This form is often described as "erysipelas migrans." In other cases the erysipelatous inflammation may attack a part far separated from the primary seat of infection. This condition may result either from infective embolism or by auto-inoculation, the cocci being carried from the primary site and being inoculated through a scratch or an abrasion into the distant part. These cases are designated "metastatic erysipelas."

If the streptococci obtain entrance to the lymphatics of the post-partum uterus, the most virulent and fatal form of puerperal sepsis results.

The ptomaïnes of the erysipelas streptococci often exert a favorable effect on neoplasms, causing destruction of tumor-cells. Cases of cure of sarcoma, carcinoma, lupus, lipoma, and keloid have been reported. This effect may be produced either by an attack of erysipelas, accidentally or purposely induced, or by subcutaneous injections of the filtered or sterilized cultures of the streptococci into or near the tumor. Attacks of erysipelas in patients suffering from diphtheria have often exerted a beneficial effect on that disease.

Complications are most commonly due to an added infection by pus microbes. There may be suppurative cellulitis or suppurative thrombo-pyelitis. Metastatic abscesses in the viscera may occur, especially in the brain and lungs. There may be septic inflammation of serous membranes, meningitis being the most important, due usually to infection through the sheaths of blood-vessels or nerves from the face. There may be ulcerative endocarditis or pericarditis. Pleurisy is not uncommon with erysipelas of the chest-wall. Pneumonia and nephritis are infrequent.

The **prognosis** is usually good except in debilitated subjects. The disease is usually fatal only by its complications.

Treatment.—*Prophylactic.*—The patient should not be attended by those who have to do with puerperal or surgical cases. The attendants must carefully protect any abrasions on their own persons, and must cleanse the hands frequently with an antiseptic solution. In hospitals the patient must be isolated and the ward and bedding be disinfected carefully. In private practice rigid isolation is not so necessary.

General treatment consists in careful nursing and feeding and in controlling annoying or threatening symptoms on general principles. Stimulants may be given freely in cases with enfeebled heart-action, and in these cases the administration of camphor in doses of gr. i–ij every hour, as recommended by Pirogoff, has been found serviceable. As a routine treatment tincture of the chloride of iron is frequently given in large doses (ʒss in glycerin and water every two to three hours), but it does not seem to exert any specific effect, and its efficacy is doubtful.

Local treatment is to be resorted to in all cases, not only to relieve the local symptoms, but also to check further advance of the disease. Hueter recommends the injection of a 2 per cent. solution of carbolic acid under the healthy skin near the advancing border. Injections of weak solutions of bichloride or biniodide of mercury may be employed in like manner. Kraske recommends multiple scarification of the skin in advance of the lesion, with subsequent moist sublimate dressings. Painting the advancing margin twice a day with a 10 per cent. solution of carbolic acid in alcohol is a simple measure, and the result is often brilliant. The spread of the disease may at times be checked by compression of the healthy skin at the periphery by adhesive straps.

To relieve the burning and itching, applications of cold water, of solutions of acetate of alumina, or of weak carbolized or sublimate solutions may be employed, but hot applications are generally more grateful and seem to exert a beneficial effect upon the disease itself. Hot lead-and-opium solution or any of the above-mentioned solutions may be employed. Ichthyol in ointment or in collodion, though recommended, is not of much service.

Abscesses and suppurations are to be treated without delay on general surgical principles.

PYÆMIA.

Definition.—Pyæmia is a septic disease characterized by repeated chills, a remittent or intermittent temperature, and metastatic abscesses in various parts of the body, due to emboli infected by the microbes of pus.

Etiology.—Pyæmia can result only from suppuration in some part of the body, the microbes of suppuration (streptococcus pyogenes and the forms of staphylococci) being necessary for its development. There must always be, in the neighborhood of the focus of suppuration, an inflammation of a vein due to the invasion of its wall by the micro-organisms. This phlebitis is regularly followed by the formation of a clot in the interior of the vein; into this clot the pus microbes make their way, as the result of which the clot becomes softened and breaks down, so that small pieces containing the microbes are swept into the general circulation until they enter some vessel too small to allow their passage. Here they will become lodged, and as the nutrition of the part suffers from the cutting off of its blood-supply by the emboli, the most favorable conditions are afforded for the development of metastatic foci of suppuration.

The infecting emboli, to get into the arterial circulation, must first pass through the lungs, which act as filters. Hence the lungs are most frequently affected. If the emboli enter the arterial circulation, they may cause abscesses in any part of the body. This is the case also in malignant endocarditis, in which disease vegetations from the diseased valves containing micro-organisms become detached, are swept into the arteries, and produce metastatic abscesses in whatever part of the body they happen to lodge. The term "arterial pyæmia" is often used to designate these cases.

Should the primary focus of suppuration occur in the district of the portal vein, multiple abscesses of the liver are produced, and there may be also suppurative pylephlebitis.

In some cases the primary suppurative focus is so slight as to be overlooked. The term "idiopathic pyæmia" is used to designate these cases. Osteomyelitis, gonorrhœa, and prostatic abscess are the conditions most frequently overlooked.

Symptoms.—The symptoms of septicæmia, and possibly the local symptoms of thrombosis of a vein near the suppurating wound, may precede the actual symptoms of the disease.

The onset is marked by a chill, which is repeated at regular or irregular intervals throughout the disease. The temperature rises rapidly during and after the chill to 103° to 105° F., and runs an intermittent or remittent course, its fall being accompanied by profuse sweating. There may be vomiting and diarrhœa. The pulse becomes rapid and feeble, and there is rapid emaciation. Delirium is infrequent, the mind usually being clear throughout the disease. The breath has a peculiar sweetish odor. There may be a septic erythema which is transitory. The face may be pale or there may be developed a moderate jaundice, usually of hæmatogenous origin. The spleen is usually large, and it may be painful and tender. There may be albumin and blood in the urine.

The symptoms of the metastatic abscesses depend upon their number, size, and locality. Abscesses in the lungs usually give rise to cough and dyspnœa.

Any of the serous membranes of the body may become secondarily affected, and septic involvement of the joints (pyæmic rheumatism) is common.

The **prognosis** is always grave. Almost all patients die in a few weeks. In rarer cases the disease may be protracted for months.

Diagnosis.—The three conditions with which this disease is apt to be confounded are malaria, typhoid fever, and acute miliary tuberculosis.

In malaria the chills, fever, and sweating occur with more regular periodicity, are checked by quinine, and in the blood the plasmodium malariæ can be found.

A careful review of the case, the occurrence of spots and abdominal symptoms, and the course of the disease distinguish it from typhoid fever.

In acute miliary tuberculosis the symptoms are more those of septicæmia: there are usually evidences of tubercular foci, and bacilli are usually present in the sputum.

Treatment consists in the aseptic treatment of all wounds, the early evacuation of abscesses if possible, and supporting the strength of the patient by feeding, nursing, and the administration of alcoholic stimulants in full doses. Quinine in large doses may be found of temporary benefit in controlling the fever, but it is of no actual service in modifying the disease itself.

SEPTICÆMIA.

Definition and Synonym.—Septicæmia is the train of symptoms resulting from the introduction into the circulation of septic micro-organisms or their ptomaïnes. *Synonym:* Sepsis.

Septicæmia occurs in two distinct forms:

1. "*Septic intoxication* is caused by the absorption of a pre-formed ferment or toxine which produces the maximum effect as soon as it reaches the circulation, and the symptoms subside with the arrest of further supply and the elimination of the septic material from the circulation.

2. "*Septic infection*, on the contrary, occurs in consequence of the introduction into the circulation of living micro-organisms which multiply with great rapidity in the blood—a circumstance which imparts to this form of septicæmia its progressive character" (Senn).

Clinical Forms.—1. FERMENTATION FEVER (Resorption fever, Aseptic fever, or After-fever) is that form of septic intoxication which results from the absorption of the products of aseptic tissue-necrosis. It follows aseptic wounds or injuries, especially if strong antiseptic solutions have been employed, causing necrosis of the superficial tissues. It may follow transfusion of blood or of simple saline solutions, and it has been produced in animals by the intravenous injection of digestive ferments. It frequently follows extravasations of blood, especially if they be confined under high tension.

Symptoms.—Within several hours after an operation or injury the temperature rises rapidly, frequently to 103° or 104° F., subsiding in from one to three days. Constitutional symptoms are slight or absent. The early occurrence of fever differentiates this from the remaining forms of sepsis.

2. SAPRÆMIA is that form of septic intoxication resulting from the absorption of the products of putrefaction. For its development three conditions are necessary: (1) *Dead tissue*, as clots in wounds or injuries, retained clots or products of conception in the uterus after abortion or labor, or tissues devitalized by traumatism, irritants, or the action of bacteria. (2) *Infection by putrefactive organisms* comprising various forms of bacteria or of the proteus groups; (3) *Sufficient time* must elapse for the generation and absorption of ptomaïnes—at least twenty-four hours.

Symptoms.—The first symptom is usually a chill, which is followed by a rise in temperature to 102° or 104° F. The pulse becomes rapid and weak, depending on the gravity of the case. Cerebral symptoms are common—headache, restlessness, and delirium. Vomiting and diarrhœa are almost constant in grave cases. The tongue is dry and often glazed, and may assume the "dried-beef" appearance. The intensity of the symptoms depends on the amount of absorbed ptomaïnes.

The *diagnosis* is rendered easy by the detection of the putrefactive focus by the sense of smell, fetor being constant.

The *prognosis* depends upon the amount of poison absorbed and upon the possibility of removing the putrefying dead tissue by surgical treatment.

3. PROGRESSIVE SEPTICÆMIA is that form of sepsis caused not only by the absorption of ptomaïnes produced at the site of the primary infection, but also of ptomaïnes produced in the blood from the microbes which it contains. The ordinary pus microbes are the most frequent causes of this form of septicæmia.

Symptoms usually arise within twenty-four hours, seldom as late as the third day. An initial chill is common. The temperature is variable: usually the fever begins gradually, reaching finally 103° or 104° F., and often it is intermittent or remittent. In some cases the temperature may be subnormal. Prostration is an early symptom. The pulse becomes rapid, weak, and compressible. There is usually mental apathy and indifference, the expression being stolid. There may be drowsiness, stupor, or delirium. The face is pale or of a yellowish tinge. Vomiting and diarrhœa are marked in severe cases. The tongue is dry, red at the edges, black on the dorsum. In severe cases there may be capillary hemorrhages.

The outlook is serious, depending upon the severity of the infection and the possibility of an early disinfection of the primary point of infection, preventing further ingress of microbes into the circulation.

Fatal cases rarely last longer than one week, and in most severe forms death may ensue within twenty-four hours.

CHOLERA.

Definition and Synonym.—Cholera is an acute infectious disease caused by the comma bacillus and characterized by purging and collapse. *Synonym:* Asiatic cholera.

Etiology.—The disease is endemic in India, a large number of cases occurring every year, especially at the places of pilgrimage along the banks of the Ganges and Brahmaputra Rivers. From time to time it makes epidemic excursions to all civilized countries, where, after remaining a certain period, the epidemic ceases and the disease is found again only in India. Thus in 1884–86 it spread from India to Egypt, Italy, Spain, the south of France, Paris, Hungary, and the Argentine Republic; it then disappeared, reappearing in 1892 in Persia, whence it had been carried from India by Mohammedan pilgrims, spreading rapidly to Russia, Germany, France, and Italy. A few cases brought on German and Italian steamers were seen in New York, but protection was afforded by strict disinfection and quarantine.

Epidemics spread along lines of travel, the infection being conveyed not only by those suffering from the disease, but also by freight, rags, bedding, etc. which have become contaminated by infected fecal discharges. The infection is not carried by the air. The epidemics travel slowly, and they can be prevented by efficient quarantine and disinfection. They are favored by poor hygiene, especially by imperfection of the water-supply, by dirty habits, and by the crowding together of people. Any condition leading to diarrhœa predisposes to infection, and for this reason the epidemics regularly are more severe in warm seasons. Cold weather may modify an epidemic or check it temporarily, but does not entirely eradicate it.

The exciting cause is a bacillus, the "comma bacillus" or the "bacillus of Koch." This bacillus is one-half the length of the tubercle bacillus, and of a curved or letter S shape. It is really a spirillum. It can readily be cultivated and can reproduce the disease. It is killed by boiling, by drying, and by acids, but it is not destroyed by freezing. It occurs in the intestinal discharges (though not when the stools become normal, nor in the diarrhœa of convalescence), occasionally in the vomita, in the intestines (whence it penetrates into the intestinal lymphatics), and in the submucosa.

The disease is not personally contagious, as it can occur only when the germ finds access to the intestinal tract. This entrance is accomplished through contaminated water or food. Nurses or washerwomen who deal with soiled bed-linen and who are uncleanly as to their hands may contaminate their food in this way.

Pathology.—There are no characteristic lesions except the presence of the bacilli. Bacteriological examinations should therefore be made in all doubtful cases. The body remains warm for a considerable time, and there is a marked post-mortem rise in temperature. Rigor mortis develops early and is well marked. There may be post-mortem movements of the body, arms, or legs. All the tissues are dry and anæmic; the blood is dark and thick; the serous membranes are sticky, dry, and may be coated with fibrin. The mucous membrane of the stomach and intestines may appear normal or sodden and œdematous, or may show traces of catarrhal inflammation. There may be croupous colitis. The intestinal glands are usually swollen. The intestines contain the rice-water discharge or a dark bloody fluid. The liver is anæmic; its cells may show parenchymatous degeneration. The kidney is the seat of an acute degeneration which is well marked. The spleen, as a rule, is not enlarged. The lungs are collapsed, and may be congested at the bases.

In patients who die in the stage of reaction the tissues are not so dry. The inflammatory changes in the lungs, stomach, and intestines, and the degeneration of the liver and kidneys, are more evident.

Incubation is usually between two and three days.

Symptoms.—The disease is usually described as occurring in the following five stages, although any of these stages may be omitted: 1. Stage of preliminary diarrhœa; 2. Stage of purging; 3. Stage of collapse; 4. Stage of warmth; and 5. Stage of reaction.

1. *Stage of Preliminary Diarrhœa.*—There is a diarrhœa with copious stools, attended with prostration and usually with nausea and vomiting. The stools are as dangerous in spreading the disease as those of the later stages. It is important to recognize these cases early, as they are frequently amenable to treatment if it be not too long delayed. This diarrhœa may continue for from one to five days, and may either end in recovery or may pass into the second stage.

2. *Stage of Purging.*—With or without this stage of preliminary diarrhœa the second stage begins abruptly, usually at night. The first symptom is copious purging, first of fecal passages, then of a frothy serous fluid without odor, containing whitish flakes of desquamated intestinal epithelium, to which appearance the name "rice-water" is applied. In some cases the discharges may be stained by blood. They may be accompanied by pain and griping, but usually their passage is painless, the fluid being voided in gushes or in an almost continuous stream.

In rapidly fatal cases the patient may die before any purging occurs. To these cases the term "cholera sicca" has been applied. Post-mortem examination shows the intestines to be full of the discharge, which has not been voided by reason of paralysis of the intestinal wall.

Vomiting regularly follows the onset of purging, the vomited matters consisting of the stomach-contents, and later of the "rice-water" fluid. There are rapidly-developed symptoms due to the loss of water from the system. There is rapid emaciation; the skin is loose and wrinkled; the eyes are sunken; the tongue is dry. The urine is diminished, and contains urea in excess, albumin, and casts; it may be suppressed. There is distressing thirst, but whatever drink is taken is immediately rejected by the stomach. There are in the abdomen and legs muscular cramps which are often agonizing. The pulse becomes rapid and feeble; respirations are shallow and sighing. The skin is cold and clammy, but the internal temperature is elevated. The mind is anxious and distressed, but is unclouded. This stage lasts for from two to sixteen hours, and from it the patients may recover or may pass into the third stage.

3. *Stage of Collapse, or Algid Stage, or Cholera Asphyxia.*—The vomiting and purging continue but become less profuse, and may finally cease. The symptoms of collapse and heart failure increase rapidly. The skin is cold and shrunken and of a leaden hue; the pulse is more rapid and thready, becoming finally imperceptible. The external temperature is low; the internal temperature is somewhat elevated. This stage lasts for several hours or it may be protracted for a day or so, and it is apt to be fatal, although patients may pass into the warm stage, and die in that, or into the stage of reaction, and recover.

4. *Warm or Tepid Stage.*—This stage is sometimes observed following the stage of collapse in patients about to die. The internal temperature rises; the skin becomes warm and of a more natural color. The purging and vomiting cease. The radial pulse can again be felt. The patient, however, becomes comatose, the urine is suppressed, and complicating inflammations of the pia mater, lungs, stomach, or intestines, or multiple abscesses, may develop. This stage is almost certainly fatal.

5. *Stage of Reaction.*—The patient may pass into this from any stage of the disease, though but rarely from the stage of warmth. The symptoms rapidly disappear, and convalescence becomes established unless interrupted by complications. There may be seen a mottling of the skin or an erythematous rash.

Complications and Sequelæ.—There is frequently suppression of urine that may persist during the stage of reaction. The patient becomes delirious, the pulse becomes rapid and feeble, and death occurs with coma. To these cases the term "cholera typhoid" has been applied. The same symptoms may be produced by septic absorption by the denuded intestinal surface.

Convalescence may be protracted by the continuance of gastro-enteritis, shown by irritability of stomach, pain, and diarrhœa. If croupous colitis occurs, it is apt to be fatal. There may be broncho-pneumonia or an irregular form of lobar pneumonia. There may be inflammation of any of the serous membranes in the long-continued cases. No symptoms, as a rule, result, and the occurrence of the lesion is known only from post-mortem evidence. There may be multiple abscesses or furuncles, frequently involving the parotid glands. There may be a continuance of the muscular spasms.

"Cholerine" is a term frequently employed by French and German authors. By French writers it is applied to cases of the preliminary diarrhœa, by German writers it designates cases of cholera running a mild course and terminating in recovery.

The **prognosis** varies from 20 to 80 per cent. according to the epidemic. The cases with preliminary diarrhœa most usually recover, especially if treated early. The prognosis in the stage of collapse is bad; in the tepid stage, almost hopeless.

Treatment.—*Prophylaxis.*—Entrance of imported cases into a port should be prevented by rigid quarantine. All merchandise and fomites should be steamed thoroughly. If quarantine and the disinfection of imported articles can be enforced thoroughly, spread of the disease should not occur.

All discharges from cholera patients should be disinfected, and all contamination of drinking-water scrupulously avoided. Those in attendance on the sick should wash the hands frequently in an acid disinfectant solution.

Health inspectors should visit the poorer parts of the infected cities to stop any cases of diarrhœa and to treat all digestive disturbances that may occur. All water should be boiled before use.

Attempts to procure immunity in man have not yet been successful.

Medicinal Treatment.—Cases of preliminary diarrhœa should be treated promptly by regulation of the diet and by the use of astringents combined with opium. As the bacillus is killed by acids, any of the mineral acids may be given; salol also is recommended. The patient should be kept quiet and cool. When the disease has once developed, treatment is directed toward mitigating the symptoms and preventing collapse. Warm applications and turpentine stupes to the abdomen and extremities, and hot baths, are frequently grateful.

The vomiting and purging are best treated by hypodermic injections of morphine, large doses being usually borne well. Calomel in a single dose of 20 grains placed dry on the tongue has at times been useful.

The thirst may be relieved by cracked ice, champagne, or acidulated water.

Mineral acids and intestinal disinfectants, such as salol, salicylate of bismuth, and β-naphthol, should theoretically be of service, but their administration is difficult because of the vomiting.

To combat the dryness of the tissues and the concentration of the blood, subcutaneous or intravenous injections of warm sterilized saline solutions should be employed, and they are often of the utmost value. Rectal injections may also be given when the purging is subsiding. Cantani recommends during the disease frequent high rectal injections of the following solution, with the hips elevated:

℞. Tannic acid,	ʒij;
Laudanum,	ʒss;
Water,	Oij.

The treatment by copious rectal enemata is termed "enteroclysis."

Heart failure is to be treated by hot applications and by hypodermic stimulation by ether, strychnine, digitalis, or whiskey. Inhalations of ammonia, amyl nitrite, or pure oxygen may be employed.

During convalescence diuretics may be indicated. The diet should be regulated, and residual gastro-intestinal symptoms should be treated as they may arise.

YELLOW FEVER.

Definition.—Yellow fever is an acute infectious disease; endemic in certain warm climates, and characterized by fever, jaundice, and a tendency to hemorrhages.

Etiology.—The disease is endemic in certain localities, principally in the West Indian Islands, parts of the coast of the Gulf of Mexico, and the west coast of Africa. It is unknown in Europe. From time to time it makes epidemic excursions into warm surrounding countries, and especially into the Southern United States. It may penetrate into more northern cities to a slight degree in the summer months. The epidemics are favored by poor hygiene and the crowding together of people and by a temperature of over 70° F. Lower temperatures diminish, and frost stops, the epidemic. Epidemics are more common on the seacoast and in low altitudes, an elevation of one thousand feet procuring nearly absolute safety. The spread of the disease is often checked by houses, walls, or streams.

Immunity may be secured by a previous attack or by long residence in an endemic locality. Native races, for the latter reason, are less liable to the disease, and when they are attacked the disease in them runs a mild course.

Yellow fever is not contagious by contact with either the living or the dead body, but the germ is cast off, in some unknown way, in an immature state, and requires further development, after which infection can occur through the medium of the air or by fomites. The disease is introduced into cities chiefly by freight and merchandise, and quarantine and disinfection are successful if thoroughly performed.

The specific germ has not yet been determined definitely.

Pathology.—The tissues of the body are usually intensely jaundiced. There may be subcutaneous hemorrhages. The heart-muscle is pale and degenerated. The liver shows advanced fatty degeneration of its cells and is of a "café au lait" color. The spleen is not enlarged. The kidneys show the lesions of either acute diffuse inflammation or a severe form of parenchymatous degeneration. The mucous membrane of the stomach is swollen and shows traces of a catarrhal inflammation, and there is frequently found in the stomach the "black vomit," which consists of mucus and altered blood.

Incubation varies from one to twenty-five days, the average duration being seven days; it is less than this in severe cases.

Symptoms.—The disease consists in severe cases of three stages: (1) stage of invasion; (2) stage of remission or "stage of calm;" (3) stage of relapse.

Stage of Invasion.—The onset is sudden and is initiated by a chill, or by convulsions in the case of children. The temperature rises rapidly to 103° F. in very mild cases, to 105° or 106° F. in the severer forms, and it may be even higher. The pulse is usually full and ranges from 80 to 120, being usually less than would be expected from the height of the fever. In some cases the pulse is slowed from the first (50 to 60). Headache and pains in the back and in the bones are decided and severe; the face is flushed; the eyes are suffused and watery. The stomach is irritable; sharp attacks of vomiting occur frequently, and may be of the projectile type. There may be vomiting of blood. The bowels are constipated. There are restlessness and possibly delirium or active mania. The urine is diminished and contains albumin and casts.

While this is the usual course, mild cases are frequently seen. The patient may be walking about with a little fever (101° to 102° F.), headache, lassitude, and occasionally vomiting. The first stage usually lasts for three days, though it may terminate in from one to six days.

The second stage, or the stage of calm, is marked by a fall of temperature, frequently to subnormal, and by disappearance of the other symptoms. In mild cases convalescence begins at this time. In these cases, as there is no jaundice, the term "yellow" fever is evidently a misnomer. In some patients who recover, however, slight jaundice may appear on the fifth day. In severer cases, after the stage of calm has lasted for two days the patient develops the symptoms of the third stage.

The third stage is marked by jaundice, by a tendency to hemorrhages, and usually by uræmic symptoms. The temperature rises, and the symptoms become aggravated. The pulse is gaseous and may be abnormally slow, falling sometimes to 40 beats in the minute. Jaundice now appears, of hæmatogenous origin, and is most pronounced. Vomiting begins anew, and in a considerable number of cases the so-called "black vomit" occurs. This is not always vomited, but may be retained in the stomach. There are usually hemorrhages under the skin, forming large ecchymoses, and from any of the mucous membranes. Cerebral symptoms are usually present. The patient may become dull and stupid, or there may be delirium, frequently of the maniacal type. Fatal cases usually pass into coma. The urine, which is greatly diminished, contains large amounts of albumin and casts, and may contain blood; in severer cases it is altogether suppressed.

In this stage the majority of patients die, from collapse, from the hemorrhages, or in a "typhoid" condition. In some cases death is due to uræmia. Fatal cases usually die between the fifth and the seventh day of the disease.

Complications and sequelæ are infrequent. There may be parotiditis, furunculosis, gastritis, or diarrhœa.

Diagnosis.—The disease is chiefly to be diagnosed from malarial fever with complicating jaundice. These cases also frequently show a tendency to hemorrhage, and occur in the same locality and at the same time at which yellow fever is apt to appear. The presence of the malarial organism and the results of quinine treatment clear the diagnosis in doubtful cases.

The **prognosis** depends upon the nature of the epidemic, varying from 10 to 85 per cent. It is better in private practice and among the native races and in young, temperate individuals. Cerebral symptoms, hemorrhages, suppression of urine, and pronounced jaundice mark the bad cases.

Treatment.—*Prophylaxis.*—There should be rigorous disinfection of all ships, merchandise, and mails arriving from infected localities. Patients should be quarantined absolutely, and their clothing and bedding be disinfected. In infected localities persons who are not necessary for the care of the sick should not remain.

The patient should be nursed carefully in a well-ventilated room, and symptoms should be treated as they arise.

Diaphoresis in the first stage often affords relief, while the headache and pains are best controlled by phenacetine, salol, opium, or single large doses of quinine.

The fever, if high, may be reduced by cold baths, sponging, the cold pack, or internal antipyretics.

Vomiting is controlled by sinapisms, dilute hydrocyanic acid, oxalate of cerium, cocaine in gr. $\frac{1}{6}$ doses, cracked ice, bismuth, or by rest and by hypodermatics of morphine.

Internal styptics have but little control over the hemorrhages.

Suppression of urine is to be treated by cups and poultices applied to the kidneys, diuretics, rectal injections of warm salt water, or the hot steam bath. Sternberg recommends small doses of bichloride of mercury with bicarbonate of soda.

The action of the heart is to be sustained by stimulants given in sufficient doses to avert danger from collapse.

SYPHILIS.

Syphilis occurs in all civilized countries, and all races are susceptible. Among aboriginal races it becomes a formidable disease, but modern methods of treatment have greatly reduced its virulence. It is said to have been inoculated in monkeys and apes, but it is doubtful if it exists in other lower animals. *Synonym:* The pox.

Lustgarten in 1844 described a bacillus somewhat shorter than the tubercle bacillus, not occurring free, but in the cells, and found in the secretions of syphilitic sores and in some gummata and condylomata. It is almost identical in appearance with the smegma bacillus. The causative relation of this bacillus to the disease has not yet been absolutely proven. Syphilis may occur as an acquired and as a hereditary disease.

ACQUIRED SYPHILIS.

Acquired syphilis, which is the most usual form, is due to inoculation by the discharges from the earlier lesions of the disease. Ordinarily it results from sexual intercourse, through minute abrasions of the genitalia. "Extra-genital syphilis" may be caused by infected throat or dental instruments, razors, pipes, cigars, cups, eating utensils, and by kissing or by beastly practices. It may be caused by infected humanized virus in vaccination, or by wet-nurses. It is not transmitted through normal secretions. Once acquired, it is rare that the disease can be reinoculated successfully.

Periods of the Disease.—Three periods are described: (1) A *primary* stage, from the moment of infection until the outbreak of constitutional symptoms, lasting for eight to ten weeks. There are two sub-periods to this stage: (*a*) first incubation period of from fourteen to twenty-one days, terminating with the appearance of the primary sore; (*b*) second incubation period, lasting from the appearance of the primary sore until the outbreak of the constitutional symptoms. (2) A *secondary* stage, comprising the fever, exanthemata, and their complications, lasting from one to three years. (3) A *tertiary* stage, consisting of gummata, visceral syphilis, and sequelæ.

Symptoms of the Primary Stage.—The first incubation period lasts for from fourteen to twenty-one days after infection, without symptoms.

The second incubation period is initiated by the development of the chancre ("hard," "indurated," or "Hunterian" chancre), which begins as a red papule on an indurated base, grows for from two to four weeks, and then slowly ulcerates in the centre; or it may begin as an indurated, indolent sore. Suppuration and gangrene may occur as secondary processes in weakly and alcoholic subjects. The chancre appears as an indurated papule on the preputial sulcus, in the orifice of the urethra, on the edge of the labia, or on the lips. The induration is broad and shallow ("parchment induration") upon the glans penis and on the inner surface of the labia. There is but one chancre, with but rare exceptions.

Care should be taken not to mistake the inflammatory induration following caustic applications to chancroids or herpes for that of a true chancre, and the possibility of a mixed infection of chancre and chancroid must be borne in mind. Microscopically the chancre consists of a cellular infiltration with acute obliterating endarteritis of the neighboring arteries.

Painless swelling of the neighboring lymph-glands occurs from eight to fourteen days after the appearance of the chancre, the glands all over the body becoming affected, the nearest glands being enlarged first. Thus in genital syphilis the inguinal glands become enlarged in the third or fourth week after the original infection, the axillary glands in the fifth week, and the cervical glands in the seventh week. The lymphatics entering the glands may be felt often as indurated cords. Suppuration does not occur unless from some secondary pus infection.

Symptoms of the Secondary Stage.—There may be fever ushering in the second stage, or the fever may appear late in the disease. The temperature may be 101° to 103° F., or even 104° to 105° F. It is marked by remissions, and, as a rule, persists for but a few days. Exceptionally the fever may last for weeks or months, and a diagnosis from malarial fever may be difficult. In one case observed by the writer the fever lasted for over four months, notwithstanding energetic antisyphilitic treatment. The spleen in prolonged fever cases is enlarged. Pharyngitis with a sharp line of demarcation on the soft palate, and tonsillitis with superficial symmetrical ulceration, are often noted, usually at the time of fever, and headache, lassitude, and pains in the back and in the limbs are complained of.

A macular eruption usually manifests itself as the first eruption. It appears on the abdomen during the eighth week after infection; on the chest during the ninth week; on the shoulders during the tenth week; on the arms during the eleventh week; on the forearms during the twelfth week; and on the hands during the thirteenth week. This eruption is symmetrical, follows the cleft-lines of the skin, and is of a dull coppery color. Papules occur somewhat later, being rarely seen before the third month after infection. They may be small or large, occurring in a row below the line of the hair on the forehead; they constitute the *corona Venerea.* Pustules may occur as a late exanthem rarely before the fourth month. The pustules may be small and shotty, closely resembling variola, or they may be large like impetigo, or they may be changed into deep ulcerations covered by rupial crusts.

A squamous form of eruption is described, not unlike psoriasis, but less scaly. This form usually is seen on the palms of the hands and the soles of the feet.

If papules occur in regions where the skin is moist, as about the anus and vulva, they become sodden, devoid of epidermis, and show white points upon their surface. These papules are the " flat condylomata."

The eruptions appearing in mucous membranes undergo maceration and are known as mucous patches, warts, and condylomata.

Mucous patches are rounded, slightly raised, and covered with a grayish film. Their discharge is exceedingly virulent. They are usually seen on the inner side of the cheek, opposite the second molar tooth, on the under surface of the tongue, and at the angles of the mouth. They are really macerated syphilitic papules. If the papillæ in the papule hypertrophy, they constitute the condyloma or wart, according to whether or not the papules are fused.

For a detailed description of syphilitic eruptions the reader is referred to special works on dermatology.

There may be extensive thinning of the hair. Iritis may develop in from three to six months after the chancre, and may be serious. Anæmia and cachexia may be marked during the secondary stage, which lasts for from one to three years.

Symptoms of the Tertiary Stage.—This stage is marked by late syphilides, gummata, amyloid degeneration, sclerosis, and visceral disease.

Late syphilides are unsymmetrical and have a tendency to deep ulceration, leaving cicatrices. The rupial stratified crusts cover round deep ulcers. Periosteal nodes along the course of the tibiæ are commonly observed. Syphilitic periostitis is marked by severe pain, worse at night—the so-called "osteocopic pain." Tubercular and unsymmetrical serpiginous eruptions characterize the tertiary period.

Gummata may develop in the skin, the subcutaneous tissues, the muscles, or the internal organs. They may be distinctly circumscribed, or they may occur as a diffused infiltration. The nodule or gumma consists of small cells with a gelatinous basement substance. It may remain unchanged, may become absorbed, may undergo cheesy degeneration can be invested with a fibrous capsule, may be converted to fibrous tissue, or may suppurate. Gummata of the skin, of the mucous membranes, of the bones, and of the cartilages undergo ulceration and extensive sloughing. In this way the cartilages of the nose may disappear, or there may be an ozæna with necrosed bone in the nose, or the palate or the tonsils may be totally ulcerated. Cicatrices of the mucous membranes may result in stricture.

In the viscera gummata frequently undergo fibroid transformation with puckering and deformity.

Amyloid degeneration frequently follows tertiary syphilis, and degeneration-changes of a sclerotic nature are commonly observed, as in locomotor ataxia and arterio-sclerosis.

The internal forms of syphilis will be treated of in detail under the titles of the diseases of the various organs.

It is important to remember that the division into the three stages is not always sharply drawn in actual experience, symptoms of one stage overlapping and encroaching upon those of the preceding or the following period; it should be borne in mind also that by systematic judicious treatment the course of the disease can be modified materially. There are also light cases in which tertiary and even secondary symptoms may not develop, especially if the case be treated from the start.

The disease is not considered inoculable after the third year.

The **prognosis** is generally good. Visceral syphilis, especially of the brain and its membranes, may terminate fatally, or death may indirectly result by arterio-sclerosis, aneurysm, or locomotor ataxia.

Treatment.—*Prophylactic.*—Syphilitic patients should not marry until three years have elapsed since the infection. It is even better to extend this period to five years. Personal prophylaxis consists in personal purity. Governmental regulation of prostitution hardly lessens the dangers. Surgical and dental instruments should be sterilized scrupulously, and it is better for the surgeon to have two sets of instruments, one set being used exclusively for syphilitic patients. Surgeons should handle syphilitic lesions with care, especially if there be abrasions on the fingers, and after such handling should cleanse their hands in strong antiseptic solutions. Syphilitic patients with mucous patches should have their own drinking and eating utensils and their own towels, and should be prohibited from kissing. A syphilitic husband should live apart from his wife for at least two years.

Curative.—(*a*) *Treatment of the Primary Stage.*—It is of no use to excise the chancre. No regular treatment should be initiated until the macular eruption appears, making the diagnosis positive. Should sloughing or suppuration occur, it should be treated on general principles.

(*b*) *Treatment of the Secondary Stage.*—The specific drug is mercury. Its use should be begun upon the first appearance of the macular eruption, and be continued for at least two years—for the first year alone, and for the second year combined with iodide of potassium.

There are two ways of giving mercury: One way is to give it intermittently, whenever symptoms appear, ceasing its use in quiescent periods; the other way is to give it steadily in small tonic doses. The latter plan seems preferable.

Mercury may be given in the following ways: Gray powder, or hydrargyrum cum creta, gr. j, with gr. j of Dover's powder, in pill, four to six times a day; tablets of the biniodide, gr. $\frac{1}{16}$, three times a day; tablets of the protiodide, gr. $\frac{1}{5}$, three times a day. Inunctions of mercurial ointment may be used. One dram suffices for a single inunction, and one application a day suffices. The ointment should be rubbed on successive nights into the lateral aspects of the thorax and the arm on one and then on the other side, then on the sides of the abdomen, then on the inner side of either thigh. This gives six localities for application. At the end of six days the patient should take a hot bath, and then the circuit is again gone over. Mercurial inunctions are recommended when an immediate effect is desired.

Subcutaneous injections of mercury have been recommended warmly. With a sterilized needle the bichloride (gr. $\frac{1}{3}$ in ♏xx of water) or calomel (gr. j–ij in ♏xx of glycerin or of olive oil) may be injected once a week into the gluteal region. Such injections, however, are apt to be painful.

Fumigations of calomel may be employed, the patient sitting in a chair wrapped in a blanket. Under the chair is to be placed an alcohol lamp over which is a strip of tin properly supported in place, 20 grains of calomel being piled in a heap at the point where the flame touches the tin (Fig. 1). The patient goes to bed after the fumigation, still wrapped in the blanket.

During the mercurial treatment care should be taken not to push the drug to the point of salivation. Before the treatment the teeth should be put in order and the tartar be removed. During the treatment the teeth should be brushed twice a day, and gargles of a saturated solution of chlorate of potassium be ordered. Green vegetables and fruit should be avoided. Upon the first appearance of salivation the drug should be stopped for the time. Should diarrhœa follow the internal use of mercurials, small doses of Dover's powder or of opium should be given in combination. Anæmic conditions call for the simultaneous administration of iron, and proper hygiene must be enforced in every instance.

At the close of one year there seems to be an advantage in combining potassium iodide with the mercury (" mixed treatment "), as in the following prescriptions:

℞. Hydrarg. bichloridi,	gr. j;
Potassii iodidi,	ʒv;
Aquæ,	
Syr. sarsaparillæ comp.,	āā ℥ij.—M.

Sig. A teaspoonful, largely diluted, after each meal.

℞. Hydrarg. bichloridi,	gr. ij ;
Potassii iodidi,	℥j ;
Tinct. cinchon. comp.,	℥iijss ;
Aquæ,	℥ss.—M.

Sig. A teaspoonful in water after each meal.

(*c*) *Treatment of the Tertiary Stage.*—The drug *par excellence* is iodide of potassium, which has a specific action. The initial dose is from 10 to 30 grains three times a day, largely diluted in water or in milk. In certain cases the drug should be pushed to greater limits, from 3 to 4 drams being required for a single dose. This is especially the case in cerebral syphilis.

HEREDITARY SYPHILIS.

The hereditary differs from the acquired form of syphilis chiefly in the absence of the primary stage.

Etiology.—*Father Syphilitic.*—(*a*) The child may be infected directly (sperm inheritance). The nearer the procreation is to the primary lesion in the father, the greater are the chances of the child being infected. The power of transmission rarely exceeds three or four years. It is possible, however, for a syphilitic father to beget a healthy child. (*b*) The mother may be infected through the placental circulation, but, whether or not active syphilis appears, a syphilitic child cannot infect the mother after birth, a maternal immunity having been created. This is known as Colles' law.

Mother Syphilitic.—If the mother be in the active stages at the time of conception, the embryo is usually infected. If the disease be acquired a few months prior to labor, the child usually escapes. Infection of the child may be minimized by judicious antisyphilitic treatment of the mother during pregnancy. About one-third of mothers abort, and gummatous placenta or degeneration of the placental follicles may be found.

Both Parents Syphilitic.—The infant mortality from paternal transmission is 28 per cent.; from maternal transmission 60 per cent.; from both mother and father, 68 per cent. Parents with tertiary syphilis are apt to beget sickly children with a tendency to neurotic affections.

Symptoms.—If the child be born diseased, there is evident poor development and malnutrition. Bullæ (pemphigus neonatorum) are usually seen upon the hands and feet; the lips are fissured; the child snuffles; the liver and spleen are enlarged; and the epiphyses are apt to be separated. Such a child is not likely to survive.

If the child be born apparently healthy, symptoms may not develop until the fourth to the eighth week. Then the child snuffles and cutaneous lesions are apt to appear, especially about the nates. There are usually brownish-red patches with a well-defined border, or they may be papular. Indolent boils in a copper-colored base may develop. Fissures (rhagades) are apt to develop about the lips, the secretions from such fissures being intensely virulent. The resulting cicatrices are characteristic. There may be falling out of the hair. Syphilitic onychia may develop.

General glandular enlargements are not so common in the hereditary as in the acquired form of syphilis. There may be purpura hæmorrhagica neonatorum, associated usually with diseased arteries. The child is apt to show increasing malnutrition, looking like a wrinkled old man. The cry is shrill and piercing. Various cutaneous eruptions may appear, with mucous patches or with ulcerations of mucous membranes. The nose may fall in. Developmental lesions of bones may develop. There may be thinning of the bones of the skull (craniotabes) or irregular growth of bone. There may be separation or suppuration of the epiphyseal ends of bones, or formation of osteophytes on the long bones. There may be gummata of internal viscera.

The teeth are apt to be deformed, constituting the "Hutchinson teeth" (Pl. 10, Fig. 2). The upper central incisors of the permanent set are usually the ones most involved. The teeth are peg-shaped, stunted, and at the cutting edge there is a notch in which the dentine is exposed.

Interstitial keratitis is apt to occur about puberty. The cornea has a ground-glass appearance which may disappear or may lead to permanent opacity. There may be iritis. Deafness from labyrinthine disease may develop. The patient has a generally stunted appearance—undersized, and apparently much younger than he really is. There may be nodosities of the long bones from gummatous periostitis, or there may be dactylitis. For a detailed account of joint- and bone-lesions the reader is referred to works on surgery.

Treatment.—The mother should receive mixed treatment throughout pregnancy, and the treatment should be continued with the child. The treatment of the child is like that of adults, but proportionately smaller doses should be given.

The syrup of Giberts is recommended:

℞.	Hydrarg. biniodidi,	gr. ss;
	Potassii iodidi,	ʒij;
	Syr. zingiberis,	
	Aq. destillat.,	āā ℥ij.—M.

Sig. Dose, gtt. v–x for a child six months old.

As a rule, mercurials have less effect upon the gums and more effect upon the bowels in children than in adults.

ACUTE MILIARY TUBERCULOSIS.

Definition.—Acute miliary tuberculosis is an acute tubercular infection characterized by an eruption of miliary tubercles in various parts of the body, with fever, symptoms of local infection, and a fatal ending.

Etiology.—The etiology of tubercular disease is considered in detail under Tubercular Disease of the Lungs (p. 373). Miliary tuberculosis is a general infection following some local tubercular lesion, and is due to the escape of tubercle bacilli into the blood, where they find lodgement in various organs and develop tubercles.

The primary tubercular focus may be apparent or may be unsuspected. It may be local tubercular disease of the lung, of the lymph-glands, of the kidneys, or of the bones. Frequently the source is found to be tubercular bronchial glands, which may even rupture into a pulmonary vein and shower bacilli into the circulation. A primary tuberculosis of the thoracic duct has been demonstrated. Well-marked cases of miliary tuberculosis have followed the use of Koch's tuberculin given for the cure of a local tubercular inflammation.

Pathology.—Tubercles (for the detailed structure of which see Tubercular Disease of the Lungs) are found in various organs of the body. They are usually the size of a pin's head, but they may appear larger from a number being coalesced. When recent they are translucent, grayish, and contain bacilli. In older cases they may be caseous and yellow in the centre and may contain no bacilli. Tubercles may be found in the lungs, pleura, peritoneum, liver, kidneys, lymph-glands, pia mater, in the bone-marrow, and in the choroid coat of the eye. Less frequently they are found in other parts. There is no regularity about their distribution: they may be abundant in some organs and scanty or absent in others, or they may be more generally distributed. This lack of regularity gives rise to great variations in clinical types. With the tubercles are often found associated various forms of inflammatory products or ordinary granulation-tissue.

Symptoms are general and local.

General symptoms are due to toxines of the general infection. Fever is a marked feature and lasts throughout the disease. Its absence is rare. It may be intermittent at the onset, resembling malarial fever. At the height of the disease it is usually markedly remittent, though it may be continuous. An "inversive type" has been described in which the highest temperature occurs in the morning—a peculiarity rarely seen in other diseases.

The pulse is more rapid and feeble than can be accounted for by the fever, running between 110 and 130.

The breathing is rapid, sometimes as frequent as 40 to 60 to the minute, and it is characteristic for the patient not to complain of shortness of the breath.

There may be profuse sweats. These may occur after remissions of temperature, or irregularly as an evidence of sepsis.

The mental condition is usually cheerful, and the mind is bright even in long-continued cases; the patient complains only of "having fever and a little cold." This sign is highly characteristic, and is often a good point of diagnosis from typhoid fever.

In acute cases with meningitis there may be delirium which is either mild or severe and amounting to mania. Stupor or coma succeeds the delirium.

The spleen is usually large; the urine is that of fever, or it may contain albumin.

The close of the disease is characterized by the symptoms of the typhoid state.

Local symptoms depend upon the locality and extent of the tubercular deposits in the various viscera. While many organs are invaded, some are more apt to give symptoms than others, and the organ most invaded will give a leading character to the local symptoms.

If the meninges are involved, the local symptoms will be those of meningitis. It is important to remember that in children tubercular meningitis is acute miliary tuberculosis with symptoms of meningitis, while in adults it is possible for it to be a local lesion without involvement of other organs.

Involvement of the pleura gives rise to pleurisy. A double pleurisy with effusion or a hemorrhagic effusion is highly characteristic.

Involvement of the lungs can be described as occurring in three stages. There is first a fine bronchitis of the smallest tubes over both lungs, especially at the apices, and associated with dry pleurisy in patches. The physical signs are those of fine bronchitis and dry pleurisy. As the tubercles increase in number they coalesce, so that parts of the lungs, especially the apices, become consolidated, giving rise to areas of dulness, to bronchial voice and breathing, and to increased vocal fremitus. In the third stage the coalesced tubercles break down to form small cavities, so that the breathing becomes broncho-cavernous and there are gurgles. The subjective symptoms consist of cough, rapid breathing, rarely a feeling of dyspnœa, slight cyanosis, and an expectoration of muco-pus sometimes admixed with blood and containing bacilli.

Involvement of the peritoneum is shown by ascites, tympanites, constipation, and sometimes by pain. In other cases there may be no symptoms although the peritoneum be extensively involved.

Tubercles in the choroid can be detected by expert ophthalmologists.

ACUTE MILIARY TUBERCULOSIS.

PLATE II.

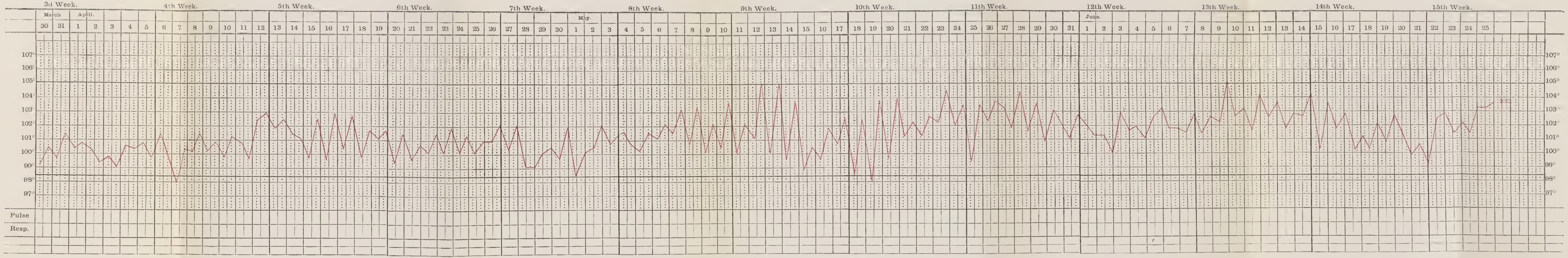


Chart of acute general miliary tuberculosis: tubercles in the pia mater, pleuræ, lungs, peritoneum, liver, kidneys, and intestines. "Inversive temperature" during the eleventh and twelfth weeks

In all cases the diagnosis is made by adding the general to the local symptoms. In some cases the general outweigh the local symptoms, and the disease resembles malarial or typhoid fever. In other cases the local symptoms are the more prominent, and the cases resemble meningitis, pleurisy, broncho-pneumonia, or peritonitis.

Types of the Disease.—1. *Fever Type.*—The disease begins like typhoid and runs for three or four weeks, with enlarged spleen. There are no hemorrhages and no spots; bronchitis is more marked, the pulse and breathing are more rapid, and the mind is clear. At the end of this time the patient may die, and the diagnosis may be difficult unless advancing lesions in the lungs and bacilli in the sputum can be demonstrated. Other cases go on for three or four months, with high remittent fever, developing areas of consolidation and breaking down in the lungs, and die exhausted. In these cases the lungs are the seat of the chief deposits. Other cases run the same course, but we have in addition the local symptoms from other organs (see Pl. 11). In still other cases the temperature at the onset is intermittent, resembling malarial fever.

2. *Meningeal Type.*—Here the picture is that of tubercular meningitis. The difference between tubercular meningitis in children and in adults has been alluded to.

3. *Mania or Delirium Type.*—Acute mania or active talkative delirium may be the first symptom, associated with fever. Stupor and coma follow, and death results, usually within three weeks.

4. *Pulmonary Type.*—There may be a pleurisy with fever out of proportion to the apparent lesion, or the disease may begin as a bronchitis or a broncho-pneumonia. Any bronchitis which persists for some weeks, with high fever, rapid pulse, and respiration with fine râles heard especially at the apices, should be regarded with suspicion.

5. Any of the preceding types may be complicated by symptoms of a pre-existing local tubercular lesion.

Diagnosis is aided by the following characteristics: In most cases there is a previous tubercular lesion. There is evidence of a diffused disease, such as meningitis, double pleurisy, and bronchitis, in the same patient. Objective symptoms are more marked than are the subjective ones. The patient "feels all right" and yet is evidently sick. In most cases bacilli can be demonstrated in the sputa and tubercles can be seen in the choroid.

There is an advancing lesion in the lung, with diffused fine râles, both bronchial and pleural; later, consolidation and breaking down in small areas.

In all cases the disease runs a progressively downward course and terminates fatally. Removal of pleuritic or ascitic accumulations is followed by a return of the effusion.

Prognosis.—The disease is surely fatal. Some cases are fatal in from seven to ten days; more commonly they run for three or four weeks or as many months, and very rarely for a year. Meningeal cases are more rapid than the fever or the pulmonary type.

Treatment is entirely symptomatic. The patient is to be fed, nursed, and made comfortable.

The sputa should be sterilized to prevent the spread of the disease.

MALARIAL FEVER.

Definition.—Malarial fever is a specific, non-contagious disease caused by the plasmodium malariæ, and is characterized by paroxysmal fever, enlargement of the spleen, and a tendency to extreme anæmia.

Etiology.—The plasmodium malariæ, discovered in 1880 by Laveran in Algiers, is not a bacterium, but is a form of protozoön or hæmatozoön. Though it has not been cultivated successfully, it is undoubtedly causative of the disease, and is of the utmost diagnostic value. It is found free in the blood and in the red blood-cells. The blood-cells become destroyed; their pigment is set free, and is found in blackish granules in the plasmodia, in the white cells, in the endothelial cells of the capillaries, and in free granules and masses. Osler describes the following six forms: (1) An unpigmented hyaline body within the red corpuscles, displaying active movements; (2) a pigmented amœboid body within the red corpuscles that may increase in size and form; (3) a segmenting body in which the protoplasm divides into a number of definite small spheres; (4) crescentic bodies which develop within the blood-corpuscles; (5) flagellate bodies; (6) the flagellæ.

The crescents are most constant in malarial cachexia. The flagellate bodies are usually found in the blood of the spleen, and they have no definite relation to any clinical form. The larger amœboid and hyaline bodies are usually associated with the acuter forms, and are readily destroyed by quinine. The flagellate bodies are difficult to see unless in motion, hence they are searched for only in fresh blood. The other varieties can be found best by rapid drying, fixation by artificial heat, with staining by a concentrated solution of methyl-blue or of gentian-violet.

The details of the life-history of the plasmodium are unknown. It is not known in what ways it enters or leaves the body, although it is supposed that infection may occur by inhaling malarial air or by drinking malarial water. The disease is not contagious.

Conditions Favoring Infection.—*Climate.*—The disease is endemic in the tropics, being less common in the temperate zones. In the tropics it occurs in severe forms with a large annual mortality. In temperate countries severe cases are rare unless they be imported. Mild cases occur often, appearing and disappearing in different years.

Season.—In the tropics cases may develop at any time, especially after the rainy season. In temperate climates the disease is chiefly one of the summer and autumn.

Age.—All ages are susceptible, but adults are more exposed to the miasm.

Sex.—The disease is more common in men because of increased exposure.

Race.—Strangers are more frequently affected than natives. Colored races are less susceptible than other races to the acute forms, but may suffer from malarial cachexia. Their immunity, however, is becoming less and less, and is often lost by a change of residence even to a non-malarial district.

Altitude.—Malarial fever is usually a disease of low altitudes, but it has been found at high elevations.

The *soil* is the breeding-place of the germ. Moisture, a temperature of 60° F. and over, and decaying vegetable matter afford the best conditions for its development. Low-lying sea-marshes and the course of rivers entering them, fresh-water marshes, especially if stagnant or if tainted with brine, damp, poorly-drained soil, and soil broken for the first time or left to waste for a number of years after cultivation, are the favorite localities for the miasm.

Degree of Exposure.—In temperate climates prolonged exposure is usually necessary; this is not the case in the tropics. Infection is more potent at night than during the day. The germ can be carried for great distances by wind blowing from infected localities.

Pathology.—There are certain lesions common to all forms. There are pigment-granules, either free in the blood or in the plasmodia, the white cells, and the endothelium of the blood-vessels. The pigmentation of the blood-vessels is more marked in some viscera than in others, being especially marked in the liver, spleen, and brain. In bad cases all the organs are pigmented grayish brown or even black.

The spleen in acute cases is congested, softened, and enlarged. It may rupture, causing fatal hemorrhage. There may be hemorrhagic infarctions in its substance. In protracted cases the spleen is large and firm, constituting a true hypertrophy.

The blood becomes anæmic from the destruction of the red cells. In protracted cases it may even resemble the blood of pernicious anæmia.

The liver during an acute attack is often congested and enlarged. Malarial hepatitis and malarial pneumonia have been described, but their occurrence has not been established satisfactorily. The presence of the plasmodia has already been alluded to.

Clinical Forms.—There are five clinical forms of malarial fever: 1. Intermittent fever; 2. Pernicious intermittent fever; 3. Remittent fever; 4. Pernicious remittent fever; 5. Malarial cachexia.

1. *Intermittent Fever* (Ague; Chills and fever).—*Symptoms.*—There may be a period of incubation of from twenty-four hours to several months. The disease occurs in paroxysms, of which three stages are described.

The *cold stage* is marked by a chill which is often severe and shaking. The skin is cold, but the internal temperature is raised frequently to 105° or 106° F. The rise of temperature usually antedates the chill. There are severe headache, and frequently nausea and vomiting. The pulse is rapid. The urine is increased in quantity. This stage lasts for from half an hour to two hours.

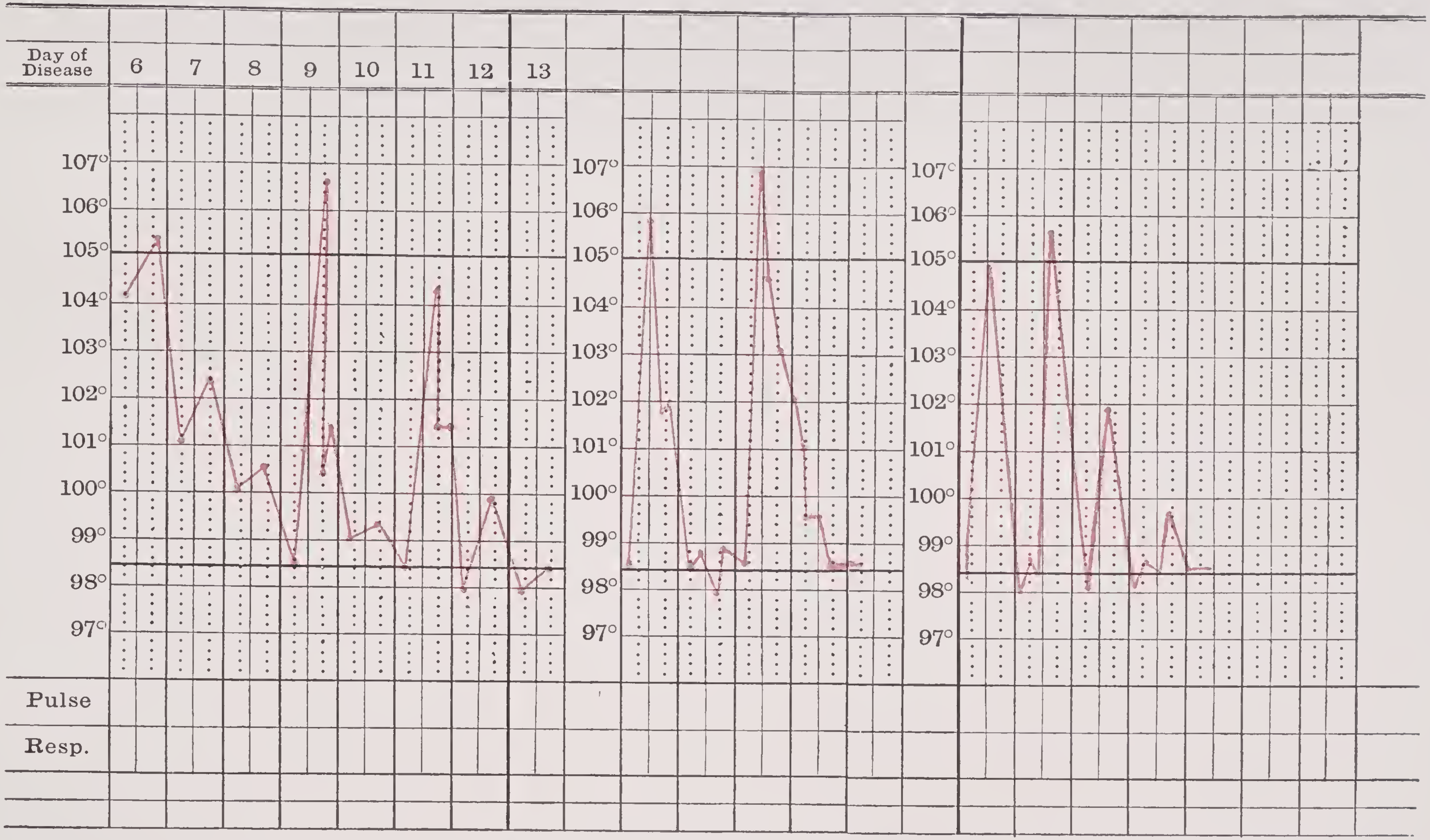
In the *hot stage* the skin becomes hot and flushed, and the fever, which may be between 102° and 107° F., becomes apparent. The pulse is full and bounding. There is a throbbing headache. There may be pains in the back and in the bones. There may be vomiting. The spleen is enlarged and tender on palpation. Examination of the blood reveals the organism. This stage lasts for from three to six hours.

The *sweating stage* is marked by the subsidence of the fever to normal or subnormal and by profuse sweating. The headache is apt to persist for some hours after the paroxysm.

The total duration of a paroxysm is between six and twelve hours. Between the paroxysms the temperature is normal and the patient complains only of lassitude. In protracted cases, however, there may be developed hypertrophy of the spleen, anæmia, and malarial cachexia. The paroxysms recur with striking periodicity, and according to the length of the intervals the principal types of malarial fever receive different names. In quotidian fever the interval is twenty-four hours, the paroxysm returning daily at the same hour. In tertian fever the interval is forty-eight hours, the paroxysm returning every other day at the same hour, which is every third day if the day of commencement of the fever be counted as the first day. In "quartan" fever the interval is seventy-two hours. Other forms are designated "quintan," "sextan," "heptan," and "octan," but they are of exceedingly rare occurrence. A "double quotidian" is a form in which two paroxysms occur in the twenty-four hours. In "double tertian" severe paroxysms occur every third day, with mild paroxysms on the intervening days.

Exceptional Cases.—In mild cases the chill is frequently absent—the so-called "dumb ague." This form occurs in the older residents of a malarial locality. In other cases the sweating stage is absent, fever and headache being the most constant symptoms.

MALARIAL FEVER.



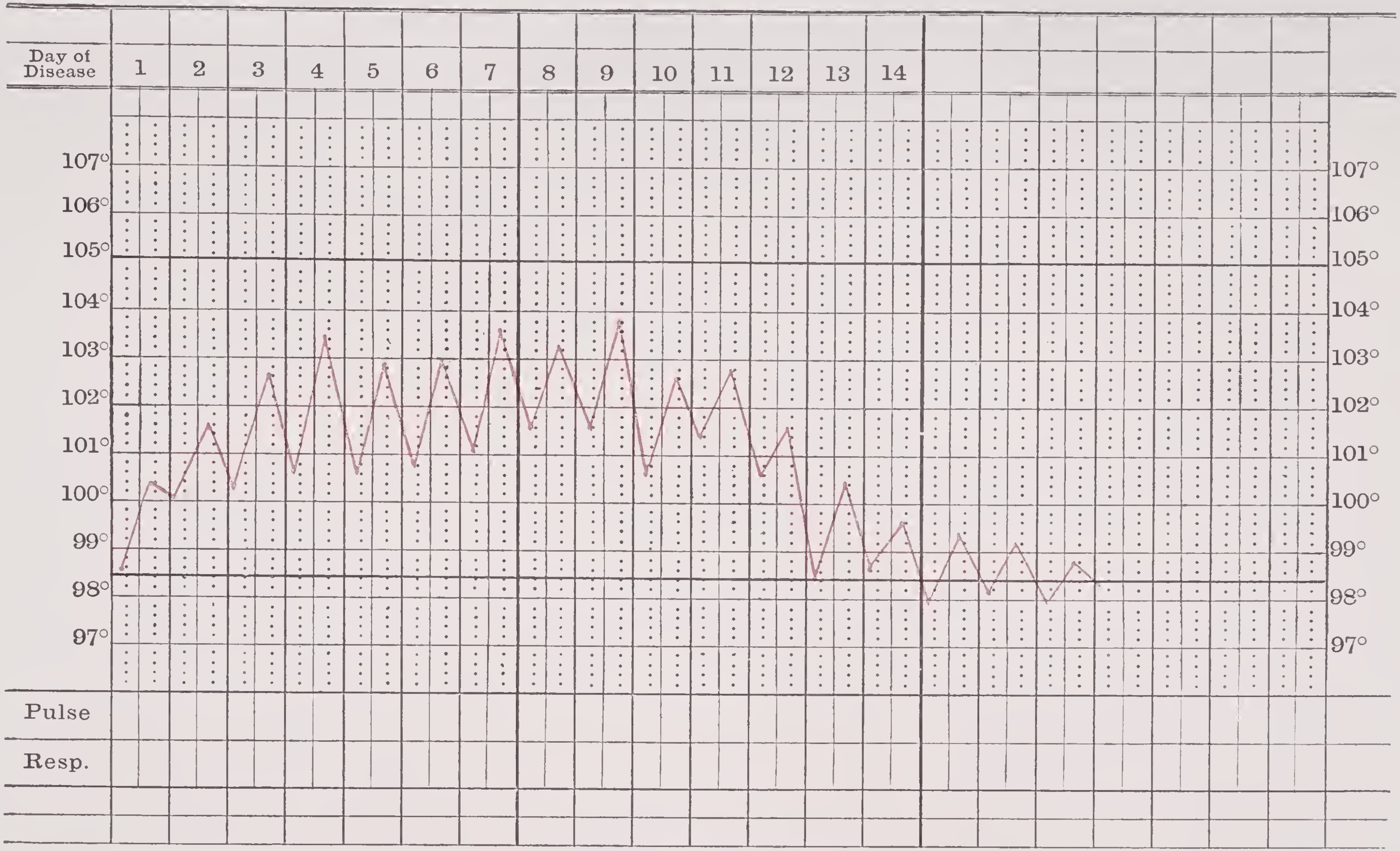
1. Tertiary intermittent fever following defervescence of lobar pneumonia.

2. Tertian intermittent fever.

3. Quotidian intermittent fever.

MALARIAL FEVER.

PLATE 13.



Temperature chart of remittent fever.

In long-continued cases the paroxysms become irregular and erratic as to both periodicity and severity.

If malarial infection occurs during the course of other disease, it gives to the original fever a greater degree of remittency.

In some patients a paroxysm may consist of a chill, fever, and the passage of urine stained with hæmoglobin.

2. *Pernicious Intermittent Fever.*—Cases of the pernicious form are but rarely seen in temperate climates unless they be imported from some intensely malarial locality. In the Northern cities of the United States cases commonly are brought from the Southern cities and from Panama. The disease is often called " Panama fever " or " Chagres fever." A number of types have been described:

(1) Some cases resemble severe forms of *ordinary intermittent fever.*

(2) Some patients pass from the paroxysm into a condition of *coma* or *collapse* in which they may die. If they recover, they are apt to die in the next paroxysm unless energetically treated by quinine.

(3) Some patients pass during a paroxysm into active *delirium or mania* which may change into coma.

(4) Some patients during the febrile paroxysm develop severe *pulmonary symptoms*, dyspnœa, cough, and expectoration which may contain blood. Examination may reveal the physical signs of a pneumonic patch or of an area of pleurisy that disappears should the patient survive the paroxysm.

(5) In other patients during the paroxysm there may be *gastric or intestinal symptoms.* There may be pain referred to the stomach, vomiting, and hæmatemesis. There may be purging resembling that of cholera, and the patient may pass blood from the bowel.

(6) Some cases are marked by a *disposition to bleed.*

Hæmoglobinuria and hæmaturia are common, but there may be hemorrhages from any of the free mucous membranes or under the skin.

Illustrative Cases.—(1) An unknown man fell unconscious in the street at 1 P. M. Temperature 97.2 F. Spleen enlarged. Plasmodia in blood. Temperature rose to 102.8° F., and the coma continued until his death at 7 P. M.

(2) A sailor just arrived from Cuba. *First day.* Became suddenly unconscious in the street. Temperature 103.2° F., pulse 92. Spleen large. Plasmodia found. Ordered quinine, gr. v every two hours hypodermically. *Second day.* Emerged from coma. Temperature normal. Feels and acts stupid. *Third day.* 2 P. M., chill, fever of 106° F., and acute mania requiring restraint, changing into coma. Quinine increased to gr. x every two hours. *Fourth day.* Entirely recovered. Had no more attacks.

(3) A sailor back two days from Panama. *First day.* Chill. Temperature 105° F.; respiration 36; pulse 112. Complete coma. Spleen large. Plasmodia found. Ordered quinine, gr. x every two hours hypodermically. *Second day.* Emerged from coma by morning, but complained of pain in the stomach; vomiting, and vomiting of blood. *Third day.* Entirely recovered.

3. *Remittent Fever.*—In this form the temperature does not at any time in the twenty-four hours become normal, but though there are marked remissions there always is a certain amount of fever. The onset may be abrupt or may be preceded by prodromal headache and malaise for several days. In some cases it follows an attack of ordinary intermittent fever. The onset is usually marked by a chill, which may be severe. The temperature, once begun, continues with remissions. Mild cases run between 101° and 103° F., severe cases between 103° and 106° F. The remissions are usually in the early morning, and they are often accom-

panied by sweating. With the fever are headache, prostration, constipation, and diminished urine of high specific gravity. During the first few days the remissions are well marked, ranging from 2° to 3°, but as the disease goes on the temperature becomes more continuous, although in milder cases the remissions may still be decided. As the disease subsides the remissions become more and more marked, and the temperature may become intermittent. In some cases there may be two daily remissions. The duration is usually from one to three weeks, and the attack may terminate in recovery or in death, or the disease may merge into intermittent fever.

Variations in Type.—(*a*) In mild cases the symptoms are not severe; the patient is up and about, and recovers in about two weeks. (*b*) In severe cases the temperature has a high range; the patient passes into a typhoid condition in which he may die in two or three weeks. (*c*) "Hemorrhagic" cases are characterized by a tendency to bleed. The most common sources of hemorrhage are the kidneys and the intestines. Considerable blood in this way may be lost. This form is usually seen only at certain times and in certain places. (*d*) In some cases there is developed well-marked jaundice, the so-called "bilious remittent fever." This fever is usually developed during the first week of the disease, and is often associated with the hemorrhagic form. The association of fever, hemorrhages, and jaundice closely resembles yellow fever, and the diagnosis is at times difficult, as both diseases are apt to occur at the same time and in the same place. Blood-examination should, however, clear the diagnosis by finding the plasmodia.

4. *Pernicious Remittent Fever.*—This type corresponds with pernicious intermittent fever, except that the temperature at no time in the twenty-four hours comes entirely to normal. No further description, therefore, is necessary.

5. *Malarial Cachexia.*—This form usually follows prolonged attacks of intermittent fever improperly treated. Cases developing without such a history are uncommon except in intensely malarial districts. The general health becomes affected; there is loss of flesh and strength; the blood shows marked anæmic changes; the complexion is muddy or lemon-colored. The spleen is distinctly enlarged and firm on palpation. The temperature may be normal or subnormal, but there is usually an irregular fever of moderate intensity. Digestive disturbances are common. There may be functional disturbance of the liver, gastric or intestinal indigestion, or irregular action of the large intestine. In other patients neuralgia is a marked symptom. Headache is the most common symptom, being frequently periodic. The next most frequent symptom is supraorbital neuralgia. The degree to which the symptoms are developed varies. In rare cases paraplegia or orchitis may develop.

Diagnosis.—The diagnosis of all forms of malaria is rendered simple by the finding of the plasmodia in the blood and by the subsidence of the disease upon the administration of quinine. The chills, the paroxysms of fever, and the sweatings of pyæmia, tuberculosis, and ulcerative endocarditis might be mistaken for malarial fever. Intermittent is to be distinguished from remittent fever by the careful use of the thermometer. Remittent fever may be mistaken for typhoid or for yellow fever. The diagnosis of the pernicious form is often difficult, but the examination of the blood, the enlargement of the spleen, the history of an intense tropical malarial exposure, and the results of quinine therapy afford positive diagnostic indications.

Prognosis.—The prognosis depends upon the severity of the infection, upon the proper treatment, and upon prolonged residence or further exposure in a malarial locality. The simple intermittent and remittent fevers do well, although if neglected they may run into malarial cachexia. The pernicious forms are exceedingly dangerous. A patient untreated rarely survives the third paroxysm. If energetically cinchonized, the prognosis is fairly good. The prognosis of malarial cachexia depends largely upon the patient's ability to change his residence to some locality where he will no longer be exposed to the miasm.

Treatment.—*Prophylaxis.*—Inhabitants of malarial districts should select the situation of their houses, should sleep, if possible, upon upper floors, and should avoid night air. It is well to take from 6 to 8 grains of quinine after breakfast.

The arrest of a paroxysm is important only in pernicious cases. A full dose of opium ($\frac{1}{3}$ grain of morphine hypodermically), $\frac{1}{5}$ grain of pilocarpine hypodermically, or a drachm dose of chloroform is often of service. Quinine is useless unless given hypodermically, as its action otherwise is too slow. In pernicious cases, however, it should be given hypodermically in full doses, the tannate, hydrochlorate, or hydrobromate of quinine being given in aqueous solution. At least 15 grains should be given every two hours in bad cases until the paroxysm yields.

During the paroxysm the chill and the fever should be treated on general principles.

To prevent recurrences quinine is a specific. It may be given in solution or in freshly-prepared capsules or pills. Children take it well in chocolate tablets. The dose depends upon the severity of the attack and the susceptibility of the patient. In mild cases from 10 to 20 grains daily will suffice. In severer cases from 20 to 40 grains will be required. It may be given in divided doses during the twenty-four hours, or in single large doses six to eight hours before the expected paroxysm. In severe cases the latter form of administration is preferable. Cinchonism may be controlled by phenacetine or sodium bromide. In many cases it is best to precede quinine treatment by a mild mercurial purgative. The effect of quinine is either to prevent paroxysms or to modify their severity or to postpone the paroxysm for several hours. A third paroxysm is not to be expected. In long-continued cases quinine seems to lose its power, and capsules of desiccated Warburg's tincture should be given. Six to eight capsules, each representing ʒj of the tincture, should be given daily. Good results frequently follow the use of arsenic, especially if combined with iron and laxatives in small doses, with or without small doses of quinine.

In remittent fever daily doses of from 15 to 20 grains of quinine frequently benefit the patient by the third or fourth day. In other cases, especially if the remissions be slight, quinine does no good, and in these cases it seems best to wait until the temperature becomes more markedly remittent, and then quinine regains its curative power. In bad cases it is best to continue the use of quinine whether it seems to do good or not.

Treatment of Malarial Cachexia.—The hardest cases to treat are those living constantly in some malarial locality. The *anæmia* demands treatment by iron and arsenic, with small doses of laxatives if needed. A good combination is the following:

℞. Ferri redacti,	gr. ij;
Pulv. ipecac.,	gr. $\frac{1}{4}$;
Acid. arseniosi,	gr. $\frac{1}{40}$;
Ext. colocynth. co.,	gr. ij.—M.

Ft. pil. No. j. Sig. Take one three times daily.

Digestive disorders are to be met with appropriate treatment. Periodical neuralgia may be benefited by quinine, arsenic, or Warburg's tincture. Obstinate cases should be sent to some non-malarial locality.

ANTHRAX.

Definition and Synonyms.—Anthrax is an infectious disease caused by the anthrax bacillus. *Synonyms:* Malignant pustule; Malignant œdema; Splenic fever; Charbon; Milzbrand; Woolsorter's disease.

Etiology.—Anthrax which is primarily a disease of cattle, sheep, and horses, is occasionally communicated to man. It is especially frequent in Russia, Siberia, in parts of Europe, and in South America. The bacillus of anthrax was the first specific micro-organism ever described. It is a rod bacillus two to ten times longer than the diameter of a red blood-cell, non-mobile, with abundant spore-growth. The rods are often jointed together, forming long filaments. The bacilli are readily destroyed, but the spores are exceedingly resistant, and live for a long time in the grass or on the surface of pasture-land. Cattle acquire the disease by eating the infected grass or by inhaling the spores.

In man the disease may be acquired by inoculation, by inhalation, or by the alimentary canal. Inoculation results from handling infected hides, wool, hair, or instruments, or by bites of flies or of mosquitoes. The disease may be acquired by inhalations from infected skins or wool, or the alimentary canal may be infected from diseased meat.

Symptoms.—The disease occurs in an external and an internal form.

1. *External Form.*—(*a*) *Malignant pustule* is the most common form, and it occurs from inoculation of an exposed surface, usually the face. Symptoms begin from a few hours to four days after inoculation, with itching, pricking, or burning like the sting of an insect. A papule is formed, developing into a vesicle which ruptures, dis-

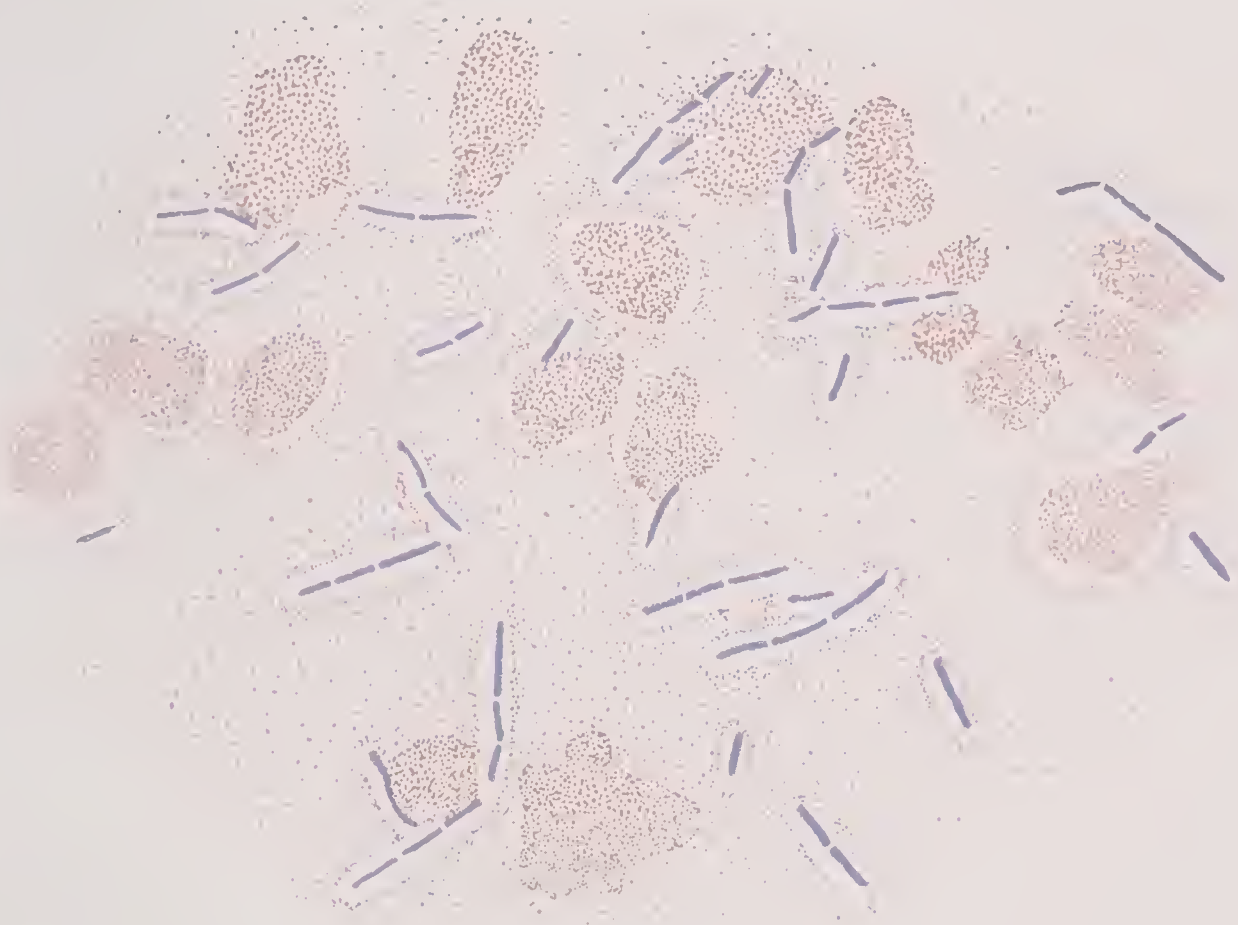
charges bloody serum, and leaves a spot of brown dry gangrene surrounded by a zone of red swollen skin which may be covered by vesicles and which resembles a carbuncle. There is brawny œdema of the subcutaneous tissues that may involve the whole of an arm or the side of the neck within thirty-six hours. There are usually lymphangitis and phlebitis starting from the infected areas.

Constitutional symptoms occur if the infection becomes general. These symptoms are usually delirium or a tranquil mind, fever, sweating, vomiting, collapse, and death, from heart failure or in the typhoid condition, in from five to eight days. If general infection does not occur, the constitutional symptoms are those which ordinarily accompany a local inflammation.

The prognosis is exceedingly grave, although in a large number of patients who recover the slough separates and the wound heals. The mortality is greatly reduced if radical treatment be resorted to at the onset.

(*b*) *Malignant Œdema.*—This form occurs in regions where the connective tissue is loose, as the eyelids, neck, and forearm. The skin may not be discolored, but there is a flat infiltration with ill-defined borders and a rapidly-spreading œdema which may be of sufficient intensity to cause gangrene. Constitutional symptoms occur early, and the disease is almost invariably fatal, although recovery is possible.

2. *Internal Form.*—(*a*) *Intestinal Form* (Mycosis Intestinalis).—This form of anthrax is caused by eating infected meat, and it may affect a number of people at the same time. The attack begins as an acute infection with a chill, fever, pain in the head and back, with severe gastro-intestinal symptoms, vomiting, and diarrhœa. There is a tendency to hemorrhages, and metastatic malignant pustules may occur on the skin. There may be delirium or convulsions. Dyspnœa and cyanosis are common, and death from heart



Bacillus anthracis: cover-glass preparation from spleen of white mouse (*American Text-Book of Surgery*).

failure occurs in a few days. These cases are invariably fatal.

(*b*) *Woolsorters' Disease.*—These cases result from sorting and picking infected hair and wool. There is a chill, followed by fever, pains in the head and chest, dyspnœa, and cough. Vomiting and diarrhœa are common. There may be a clear and tranquil mind, although delirium and unconsciousness are frequent. There is increasing heart weakness, and death results, in collapse and extreme prostration, in from one to seven days.

Treatment.—Preventive treatment should be directed to cattle and sheep. Diseased animals should be killed and buried deeply or cremated; suspected animals should be isolated. Inoculations with attenuated virus are being employed with considerable success in securing immunity.

In man the only form amenable to treatment is the external pustule. The mass should be excised thoroughly or be penetrated by deep crucial incisions into which powdered bichloride of mercury is to be sprinkled. Subcutaneous injections of a solution of carbolic acid or of a bichloride-of-mercury solution may be made about the pustule and be repeated two or three times a day.

HYDROPHOBIA.

Definition and Synonym.—Hydrophobia is an acute specific disease of animals, and is communicated to man by inoculation. *Synonym:* Rabies.

Etiology.—All animals are susceptible, but the disease is most common in the dog, wolf, cat, skunk, and fox. There is undoubtedly a microbe of the disease, but it has not yet been demonstrated. The poison is found in the nervous system and the secretions, especially in the saliva. The affection is acquired in man by bites of rabid animals or by the inoculation of abraded surfaces with the saliva. It occurs more readily in children than in adults, and infection is most certain in wounds of the face and head and in cases of severe and lacerated bites affording extensive surfaces for absorption. Infection is more severe in the bites of wolves than in those of cats or dogs. Of persons bitten by rabid dogs, from 15 to 50 per cent., according to various authorities, become affected by the disease. The infecting saliva may be absorbed by the clothing if the bite is inflicted upon a clothed part. In this case infection may not occur.

The incubation period varies from six weeks to two months in ordinary cases. It may be as short as two weeks, or be protracted for several months, but never longer than eight months. Cases with incubation of from one to two years have been reported, but they are not well authenticated.

Pathology.—Little or no lesion may be found at autopsy. Congestion of the blood-vessels, perivascular exudation of leucocytes, and minute hemorrhages may be found in the brain and spinal cord. These are the most characteristic lesions, and are particularly well marked in the medulla. There may be congestion of the pharynx and of the mucous membrane of the respiratory and gastro-intestinal tracts.

Symptoms.—There are three stages of the disease.

1. *Premonitory Stage.*—The onset is usually preceded by irritation, pain, or numbness in the cicatrix, which may become congested and tender. The patient passes into a condition of mental depression and melancholia, becomes irritable and sleepless, and is in a condition of extreme anxiety. The special senses are keenly alert. This stage lasts about a day.

2. *Spasmodic Stage.*—The first characteristic symptom is inability to swallow, from spasm of the muscles of deglutition whenever the act is attempted. The spasm spreads

to the laryngeal muscles of respiration, causing dyspnœa and the utterance of odd barking sounds. Breathing in consequence becomes painful and embarrassed. The spasms, excited at first by attempts at swallowing, finally are produced by any afferent stimulant, such as draughts of air, sounds, or even mental suggestion, and extend to involve the muscles of the body generally. During these convulsions the patient snaps with his mouth and ejects foaming saliva in every direction. Mania often accompanies the spasms, while in the intervals the mind is usually clear, though distressed by fearful dread of impending death. Profuse salivation is common. The temperature is usually elevated, although it may be subnormal. The pulse becomes increasingly rapid, feeble, and intermittent. Prostration becomes more marked after each paroxysm. There may be death from asphyxia in any of the paroxysms. This stage usually lasts for from one to three days.

3. The *paralytic stage* lasts for from six to eighteen hours. The patient becomes quiet, the spasms cease, and the patient may swallow with ease. Unconsciousness is gradually developed, and death from cardiac failure occurs.

Prognosis.—Much can be done by preventive inoculations in the stage of incubation. When the disease, however, has once begun, it is invariably fatal.

Treatment.—Immediately after a person has been bitten a ligature should be applied on the proximal side, and the wound be sucked energetically, provided there be no carious teeth or abrasions of the mouth or lips of the operator. The best results have followed an immediate excision of the wound succeeded by thorough disinfection. Cauterization is not so certain as excision as a method of prevention.

Preventive Inoculations.—Pasteur found that the continuous inoculation of the virus from rabbit to rabbit increased its strength to a maximum virulence, while gradual desiccation of the medulla containing the virus diminished its virulence so that after two weeks' desiccation the virus became innocuous. It is possible, then, to obtain the virus in any grade of virulence.

Inoculations were made in dogs, beginning with injections of an emulsion of the non-virulent medulla, followed by those of increasing strength until injections of medullas of the greatest virulence could be made with impunity. Animals so treated became immune to hydrophobic infection.

The same series of injections are now made in the case of those who have been bitten by rabid animals (although in men the final injections used are not of the most virulent quality), with the result that the occurrence of hydrophobia in nearly all cases is prevented. The mortality of those bitten by rabid animals and treated in this manner has been reduced to 0.60 per cent.

There is another method of antirabic vaccination proposed —the gradual inoculation of an innocent virus obtained by the action of gastric juice upon the cords of infected rabbits. This method has even cured the developed disease in rabbits, but it has not yet been tried in man.

Treatment of the Disease.—When hydrophobia develops the spasms can be relieved only by morphine hypodermics and by inhalations of chloroform. The patient in this way is made more comfortable until he dies.

Pseudo-rabies is an hysterical condition occurring in persons who have been bitten by dogs supposedly rabid, and it may closely simulate true hydrophobia. The *symptoms* develop frequently too long after the bite to be real rabies; the temperature is not elevated; the disease is of longer duration and is amenable to antihysterical treatment. The *diagnosis* is corroborated by the knowledge of the fact that the dog was not rabid.

TETANUS.

Definition and Synonyms.—Tetanus is an infectious disease due to a specific bacillus. It is characterized by painful tonic spasms of the voluntary muscles with exacerbations. *Synonyms:* Trismus; Lock-jaw.

Etiology.—The bacillus appears as a delicate rod swelling at one extremity to contain a shining spore, assuming thus the appearance of a drum-stick or a pin. Pure cultures are obtained with difficulty. Injections of the germs, or even of the sterilized or filtered cultures, cause tetanus convulsions. Brieger has lately isolated from tetanus cultures their distinct toxines, tetanine, tetanotoxine, and spasmotoxine, which act on the nervous centres as does strychnine, causing convulsions and spasms.

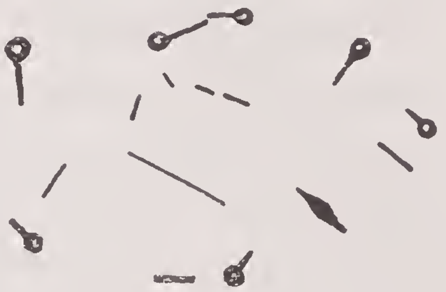


FIG. 3.—Bacillus tetani (cover-glass preparation from culture by Kitasato).

The bacilli are found in various kinds of surface soil and street dust. In warm climates the soil acts as an excellent culture medium, hence in these localities the disease is more common than in colder climates. The bacilli may thrive in some particular soil so that the disease becomes endemic in that place, as in the east end of Long Island. It may assume epidemic proportions in institutions, in hospitals, and during wars. The colored race is especially susceptible.

To acquire the disease the germ must enter through an abraded or a broken cutaneous or mucous surface. It may follow wounds, especially of the hands and feet, or it may infect the umbilicus in newly-born children (tetanus neonatorum). It frequently follows frost-gangrene. In some cases the point of entrance is so slight as readily to be overlooked. These cases are designated "idiopathic tetanus."

The bacilli are found in the wound-secretions, in the nerves leading from the point of infection, and in the spinal cord of the patient.

Pathology.—There are no distinct morbid changes. There may be seen congestion, perivascular exudation, and granular degeneration of the nerve-cells in the brain and spinal cord. There may be redness and swelling of the nerve-trunks. In tetanus of the new-born there may be inflammation of the umbilicus.

Symptoms.—The period of incubation is about two weeks, although from one to twenty-one days constitute its limits. The symptoms begin insidiously with soreness about the neck and pain and stiffness in the muscles. Then develops the characteristic spasm of the muscles of mastication. This trismus, or lock-jaw, is almost pathognomonic, provided local causes for spasm can be excluded. The spasm then spreads to the muscles of the back of the neck, of the face, and of the trunk. The head is drawn backward and held rigidly; the face has a mask-like appearance due to immobility; the corners of the mouth are drawn back, giving the "sardonic grin." The forehead is wrinkled, the patient having a peculiarly aged appearance. The spasm of the spinal muscles may arch the body backward so that the patient is supported only by the head and the heels (opisthotonos), and less frequently the body may be flexed forward (emprosthotonos) or curved to either side (pleurosthotonos). In severe cases the body is entirely straight and stiffened from general muscular spasm (orthotonos). The legs may be in a condition of spasm, but the arms more frequently can be moved freely.

The affected muscles are in a condition of tonic rigidity interrupted now and then with violent clonic spasms.

These spasms may be so severe as to tear the rectus abdominalis. The muscles are exquisitely painful and tender, especially during a paroxysm.

The paroxysms are reflex, and are produced by any slight stimulus, such as a noise, a jar, or a draught of air. In some cases they may appear to be spontaneous.

If the intercostal muscles are affected, there is embarrassed respiration with dyspnœa and cyanosis. These symptoms are so aggravated, should the diaphragm be involved, that the patient rarely survives the second or third tetanic spasm.

In rare cases the muscles of deglutition are involved. These cases are spoken of as "hydrophobic tetanus" or the "head-tetanus" of Rose, and follow injury of the face. Besides the trismus and the difficult deglutition there is apt to be paralysis of the facial nerve on the side of the injury.

The temperature is always elevated in acute cases, usually running to 104° F. or even higher. There may be a marked rise of temperature after death. In the milder and more chronic cases there may be but slight fever or the temperature may be normal. The pulse becomes rapid and feeble toward the close of the disease. The mind is clear throughout. There is usually profuse perspiration.

The **duration** varies. The acute forms may be fatal in from one to seven days. If the attack be mild, there may be but little spasm of the muscles of the trunk, and constitutional disturbances are not marked. In these cases trismus is the principal symptom. These mild cases run a course for a number of weeks.

Diagnosis.—Tetanus should not be confounded with the following conditions:

1. *Strychnine-poisoning.*—Here the maximum symptoms are developed suddenly, the muscles not being involved in gradual order as in tetanus. Trismus is absent and the

arms are involved. Between the spasms the muscles are relaxed, and not rigid as in tetanus. The course is shorter than that of tetanus.

2. *Hydrophobia.*—Here there are the history of the case, involvement chiefly of the muscles of deglutition, and absence of trismus.

3. *Hysteria.*—The convulsions are not limited to any special group of muscles; there is no trismus; between the spasms there is muscular relaxation; there is no change in temperature or pulse; other hysterical symptoms are present; the spasms are irregular and more spontaneous than reflex.

Prognosis.—The disease is fatal in 80 per cent. of traumatic cases and in 50 per cent. of idiopathic cases. It is almost always fatal in infants. Most of the cases of hydrophobic tetanus recover. When the incubation period is less than ten days, the mortality is 96.6 per cent.

Treatment.—The patient should be kept in a darkened room from which all sounds and other causes of irritation are to be excluded absolutely. No talking or unnecessary movements are to be permitted. If the trismus prevents the patient from taking even fluid food, he may be fed through a tube or by the rectum. To relieve the spasms morphine hypodermically is the most satisfactory drug. In milder cases chloral combined with sodium bromide may be employed. In very severe cases inhalations of chloroform may be necessary. In mild cases the use of hot baths may be of service. Woorara, which has been employed frequently, is not recommended, because of its depressing effect on the heart. In all cases the infected wound must be disinfected to prevent further absorption. Scars may be excised and foreign bodies be removed. Nerve-stretching and nerve-section have proved disappointing.

The hope of successful treatment lies in the use of antitoxines derived from the blood-serum of animals rendered immune. Immunity is procured by the injection of germ cultures treated with trichloride of iodine. The injection of the blood-serum of such immune animals into persons suffering from tetanus has frequently been followed by a prompt recovery, while in almost all cases the severity of the disease has been modified greatly.

LEPROSY.

Definition.—Leprosy is a chronic infectious disease due to the bacillus lepræ and characterized by tubercular nodules of the skin and the mucous membranes and by changes in the nerves.

Etiology.—At present the principal centres of leprosy are India, China, and the Sandwich Islands. Other important foci are Norway, the Baltic provinces of Russia, Mexico, certain parts of Central and South America, and the West India Islands. In North America it occurs in certain of the Gulf cities, especially New Orleans, in the province of New Brunswick, and along the Pacific coast, where it occurs chiefly among the Chinese. Isolated cases occur from time to time in all large cities.

The disease may be called "contagious," but only in the sense that direct inoculations are necessary, as is the case with syphilis. It may be congenital or hereditary, and it may be acquired through sexual congress. The majority of cases occur from the fifteenth to the thirtieth year. The specific cause is the bacillus lepræ, discovered in 1874 by Hansen. This bacillus, which closely resembles the tubercle bacillus but may be distinguished from it, can be cultivated; but while inoculations of the leprous nodules can reproduce the disease, inoculations of the pure bacillus cultures have produced only negative results.

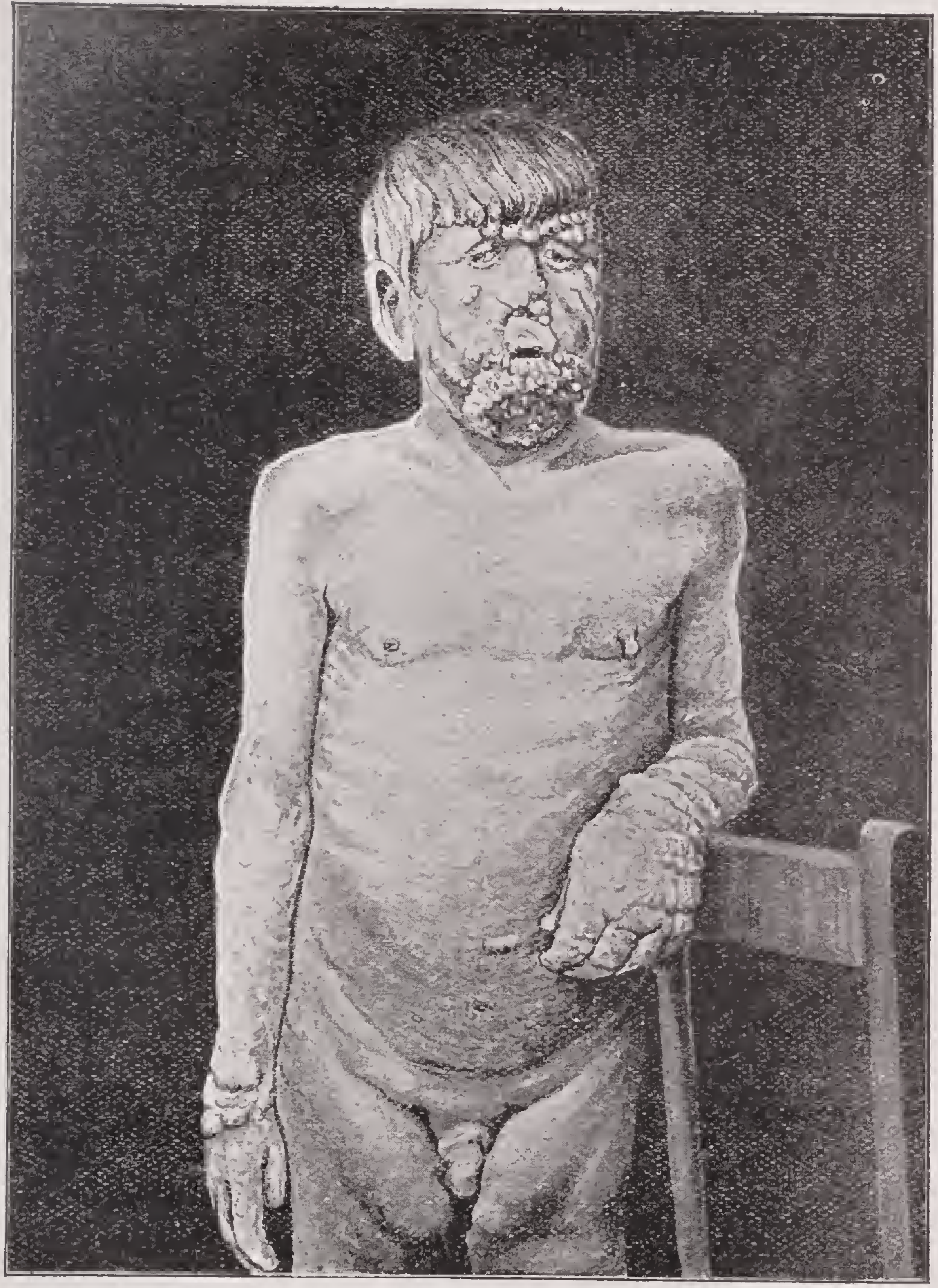
Pathology.—The leprous nodules consist of aggregated lymphoid, epithelioid, and giant cells in and among which are found numerous bacilli. The nodules may in rare cases become organized and encapsulated, but they have a tendency to break down, discharge puriform matter, and result in ulcers which may heal in one direction while spreading in another. In the nerves the bacilli cause neuritis. In the last stages of the disease leprous nodules may be found in internal organs, especially the spleen and the liver.

Symptoms.—Two forms are described, which may occur separately or be combined in the same patient:

1. *Tubercular Leprosy.*—There appear on the skin hyperæsthetic patches of sharply-defined erythema that become gradually darker from pigmentation. These patches which precede the nodules are designated as "macular leprosy." In some cases they subsequently become anæsthetic and lose their pigment, white spots being left, the "white leprosy." Nodules then develop in the skin of any part of the body excepting the scalp, and in the mucous membranes, especially of the mouth, throat, larynx, and conjunctiva. The nodules vary in size from a pea to a walnut, and coalesce. This is especially marked in the face, the term *leontiasis* being applied to the thickened and distorted features so caused.

The skin over the nodules is tense and glistening and may become red and painful. The hairs of the affected areas drop out, the loss of the eyebrows being a suggestive symptom. From the softening and breaking down of the nodules there are caused extensive ulcerations frequently covered with crusts. The ulceration may extend to the bones, causing falling of the bridge of the nose or loss of the fingers or toes, or there may be total destruction of the eyeball.

2. *Anæsthetic leprosy* occurs when the nerve-trunks are involved. There are at first areas of hyperæsthesia and



Tubercular leprosy in a patient twenty years old (Goldschmidt).

neuralgic pains, followed by anæsthesia over more or less extensive surfaces. The anæsthetic spots following the macules have already been alluded to. If the larger nerve-trunks are involved, they may be felt as nodular cords. Suppression of sweating occurs in the affected areas.

There are trophic changes. Bullæ may form at any time, and after discharging their contents may either heal or be converted to extensive ulcers. There may be perforating ulcer of the foot or loss of the phalanges of the fingers or the toes. Paralyses, contractures, and atrophy of muscles are commonly observed.

The **prognosis** is bad, but not absolutely hopeless. The average duration of the tubercular cases is from eight to ten years; of the anæsthetic cases, fifteen to twenty years.

Treatment.—Patients should live in isolated communities and under the best hygiene. Their general health and nutrition must be superintended carefully.

There is no specific medication. Iodide of potassium in full doses (falling short, however, of iodism) and arsenic have been recommended. Of late gurjun oil in 10-minim doses and chaulmoogra oil in 2-dram doses have been favorably regarded. The former may be given by the mouth or by inunction.

GLANDERS.

Definition and Synonym.—Glanders is an infectious disease of the horse, ass, and mule, communicable to man. *Synonym:* Farcy.

Etiology.—The disease is due to a specific bacillus, the bacillus mallei, which is short and non-motile, closely resembling the tubercle bacillus. The bacillus can be cultivated, and causes the disease by inoculation. The disease is acquired in man by contact of the nasal discharges of the horse with an abraded mucous or cutaneous surface. In rare cases it results from inhalation of the desiccated discharges. The disease is also transmissible from man to man. It occurs chiefly in those who have to do with horses.

Lesion.—The lesion consists in the formation of little tumors composed of epithelioid and lymphoid cells among and in which are found the bacilli. The lesion is really a variety of infective granuloma. The nodules tend to break down rapidly, causing ulcers when they occur in the mucous membranes (glanders), and abscesses when they occur in the skin, the muscles, or the internal viscera. The adjacent skin, lymphatics, and mucous membrane are inflamed.

Symptoms.—The symptoms begin in from three to five days after inoculation, but may be delayed for three weeks. An acute and a chronic form may be recognized in man.

1. *Acute Glanders.*—There are at the onset malaise, headache, fever, and pain in the limbs, resembling the onset of typhoid fever, for which it is often mistaken. In peracute cases the onset is that of a more severe general infection. The local symptoms may be of the "farcy" or of the "glanders" type.

Farcy Type.—The infected part becomes red, swollen, and painful, the inflammation becoming widely diffused; there are developed nodules which become abscesses. These latter may rupture, leaving irregular deep ulcers with a grayish infiltrated floor which may become necrotic. These suppurating nodules are frequently mistaken for small-pox. The lymphatics are early affected, and along their course are subcutaneous nodules, the so-called "farcy-buds." There may be swelling and suppuration of the joints. Abscesses may form in the muscles.

Glanders Type.—There is a purulent, blood-stained, fetid discharge from the nose, with a spreading inflammation of the skin over the nose and the face somewhat resembling erysipelas. Examination shows the nasal cavities to be deeply ulcerated. The septum may be necrosed. There may be similar ulcerations in the mouth, pharynx, larynx, and bronchi. Usually in man both sets of local symptoms are found.

Constitutional symptoms are those of an intense infection. The temperature rises and may assume the pyæmic type, with remissions and irregular rises accompanied by chills and sweating. The pulse becomes rapid and steady, the tongue brown and dry. Vomiting and diarrhœa are almost constant. Pneumonia is apt to develop. There are restlessness and delirium at first, passing into stupor and coma, while death results in the typhoid condition in from eight to fourteen days. Peracute cases may live but a few days. In mild cases the local symptoms are less severe and the general infection is slight and limited, so that the course is mild and recovery is possible. The cases in which the nose is extensively involved—glanders type—are invariably severe and fatal.

2. *Chronic Glanders.*—The disease is insidious, resembling ozæna or nasal syphilis. There is a fetid, purulent discharge from the nose, with intractable ulcerations. There are also subcutaneous nodules, abscesses, and ulcers without much inflammatory reaction or involvement of the lymphatics. These cases may last for months or even years, and usually recover, although at any time the acute form may be developed with a fatal issue.

Treatment.—In the early stages the wound should be excised or cauterized and treated antiseptically. For the acute cases little can be done. The nasal passages should be kept cleansed by injections of weak antiseptic solutions. Abscesses and farcy-buds should be opened as early as possible.

ACTINOMYCOSIS.

Definition.—Actinomycosis is a form of chronic inflammation caused by the actinomyces, or ray fungus.

Etiology.—Actinomycosis is a disease primarily of cattle, pigs, or horses, acquired in man by inoculations through abrasions of the skin, of the mucous membrane of the mouth, or through carious teeth. The fungus may be taken into the alimentary canal in contaminated water, in the flesh of the pig, or in infected cereals.

The ray fungus, or actinomyces, consists of threads with bulbous extremities radiating from a common centre, forming a globular rosette. In man the fungi appear as little round masses the size of a millet-seed, usually of a sulphur-yellow color. They are found in the tumors and in the purulent discharges, and can be cultivated and inoculated.

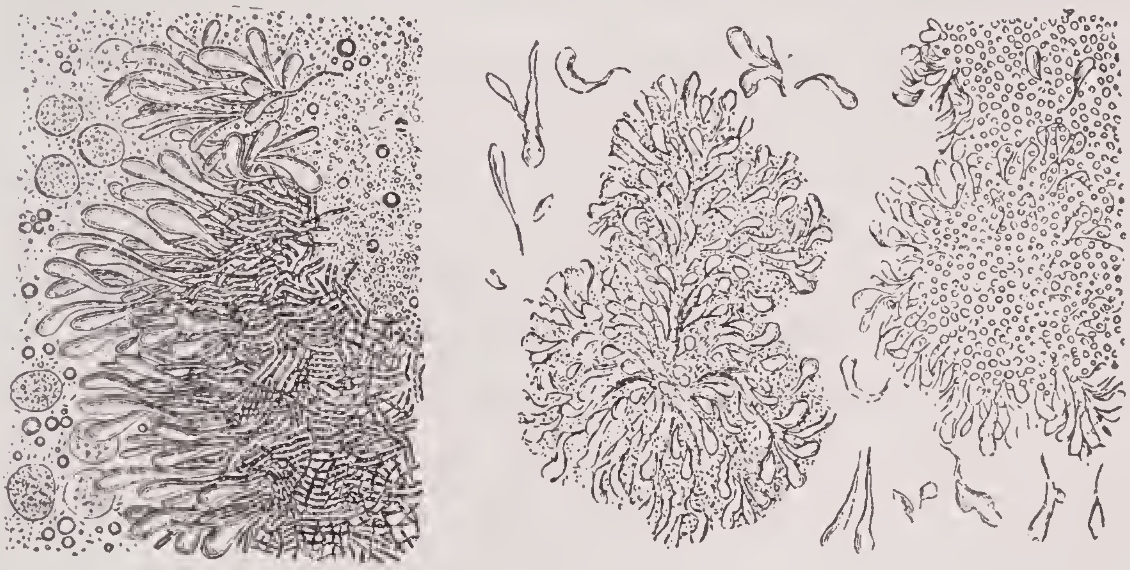


FIG. 4.—Actinomyces (Von Jaksch).

Pathology.—The lesion consists in the transformation of mature connective tissue into embryonal or granulation-tissue composed of round and epithelioid cells sometimes containing giant-cells. The appearances are identical with those of sarcoma or tubercle, but in the tumors the characteristic ray fungus is found. The tumors show a tendency to break down and suppurate, forming abscesses and sinuous fistulæ. There is a chronic inflammation of the surrounding structures, but the lymphatics are not involved, and the course of the disease resembles that of a malignant tumor more than that of an acute infection.

Symptoms.—1. *Alimentary Actinomycosis.*—The upper or lower jaw is frequently involved. There is considerable swelling and enlargement of the bone resembling osteosarcoma; sinuses are common, tending to invade the adjacent soft structures of the neck and the face. The tongue, the intestines, or the liver may be involved primarily or by metastasis.

2. *Cutaneous Actinomycosis.*—There may be slowly-growing tumors which may suppurate and leave intractable ulcers and fistulous tracts.

3. *Cerebral Actinomycosis.*—This form is rare. The symptoms are those of multiple tumors or abscesses.

4. *Pulmonary Actinomycosis.*—Three clinical forms are described by Hodenpyl: (*a*) There may be the lesions of chronic bronchitis with the actinomyces in the sputum. (*b*) Small nodules may be scattered in the lungs, resembling miliary tuberculosis. (*c*) The lesions may be more extensive—broncho-pneumonia, interstitial pneumonia, large abscesses. In some cases there may be abscesses of the chest-wall that may be mistaken for empyema.

The **prognosis** is good if the disease be recognized early and if the infected locality be amenable to surgical treatment. Spontaneous recovery does not occur. The prognosis of internal actinomycosis is bad.

Treatment is entirely surgical, consisting in the excision or cauterization of the diseased masses and in opening and disinfecting abscesses and sinuses. Injections of weak antiseptics into diseased internal tissues may be made.

MILK SICKNESS.

This disease occurs west of the Alleghany Mountains and is associated with the "trembles" in cattle. It is communicated to man by infected meat or milk, also by butter and cheese. The animals subject to the disease are cattle, lambs, and colts. The disease is most frequent in new settlements, and with the advance in civilization is rapidly becoming extinct.

The **pathology** is unknown.

The **symptoms** *in cattle* are refusal of all food, injected eyes, marked tremors or "trembles," and death in convulsions.

The symptoms *in man* begin with abdominal pain, nausea, and vomiting. There is intense thirst with fever. The breath becomes peculiarly offensive. The tongue is swollen and tremulous. Cerebral symptoms develop—restlessness, delirium, and coma; the patient passes into the typhoid state and dies. Acute cases last for two or three days; subacute cases continue for two or three weeks.

The **treatment** is entirely symptomatic.

WEIL'S DISEASE.

This disease was first described in 1886 by Weil, and bears his name. The exact nature of the disease is unknown. It is most common in the summer months, when small epidemics may appear. It attacks males more frequently than females, is most common between the ages of twenty-five and forty, and butchers seem especially liable to it.

Symptoms.—The onset is acute, being marked by headache, by pain in the back and muscles, and by fever which is usually of a pronounced remittent type. Jaundice appears early and is of the obstructive form, with clay-covered stools.

The liver and spleen are swollen, and the liver is tender on palpation. Albuminuria is commonly observed. Gastro-intestinal symptoms are but rarely observed. Cerebral symptoms may appear in severe cases. The duration of the disease is from ten to fourteen days.

II. DISEASES OF THE CIRCULATORY SYSTEM.

I. DISEASES OF THE PERICARDIUM.

THE diseases of the pericardium may be described anatomically and clinically under the following divisions: 1. Acute plastic or dry pericarditis; 2. Pericarditis with effusion; 3. Purulent pericarditis, or empyema of the pericardium; 4. Chronic adhesive pericarditis; 5. Tubercular pericarditis; 6. Cancerous pericarditis.

Dry pericarditis and pericarditis with effusion may be considered together under the general heading of

PERICARDITIS.

Etiology.—Pericarditis is due to infective processes or to the extension of inflammation from contiguous organs.

Primary or *idiopathic* cases are rare except as the result of traumatism either external or from within, as by the passage of a foreign body through the œsophagus to the pericardium.

Secondary inflammation is common, from the following causes:

(*a*) Pericarditis from extension may complicate various diseases of the lung or the pleura, such as pneumonia, pleurisy, tubercular disease, or cancer. In double pleurisy the pericardium is usually involved. The disease may result from the extension of inflammation of the abdominal organs, such as peritonitis or abscess of the liver; it may also result from inflammation of the heart-wall, as in myocarditis.

(*b*) Pericarditis is one of the most important complications of rheumatism, occurring in from 14 to 37 per cent. of all cases of the latter disease. The disease may occur with either mild or severe forms, may precede the articular affections, and often occurs in the abarticular forms of rheumatism in children.

(*c*) Pericarditis may accompany any of the eruptive fevers, especially scarlatina.

(*d*) The disease may complicate any of the septic diseases, as pyæmia, puerperal fever, malignant endocarditis, septic thrombi, gonorrhœa, bone necrosis, or influenza. It is not infrequent in pneumonia.

(*e*) Certain dyscrasias render the pericardium susceptible to inflammation. Gouty, scorbutic, and diabetic patients are liable to pericarditis, while with Bright's disease pericarditis is by no means rare, especially in patients over fifty years of age.

Pericarditis occurs at all ages, but is more common in young adults. It may be a disease of intra-uterine life, or it may occur in the newly-born from septic infection through the navel. In childhood pericarditis is more commonly due to rheumatism or scarlet fever, while in advanced life it is oftenest associated with the atrophic form of Bright's disease.

The lesion of pericarditis resembles that of the kindred inflammations of the pleura in which the inflammation may be plastic with but a small amount of effusion, or may pass into the stage in which effusion is abundant and alters the clinical course and the physical signs of the disease. The inflammatory process, however, is the same, differing only in the amount of serum exuded.

The lesion may be localized or general at the start. The base and the anterior surface of the heart are oftenest affected. The pericardium becomes dry, congested, lustreless, and covered with an exudate of fibrin in the meshes of which a certain amount of fluid effusion is entangled. The fibrin may be thin and dry like a membrane, under which the pericardium appears congested, or it may be in long shreds, giving a hairy appearance to the heart—the *cor villosum;* or, if the fibrin is abundant, the pericardium may have a ridge-like appearance resembling buttered surfaces of bread drawn apart—the "bread-and-butter pericardium." This is the lesion of plastic pericarditis. Pericarditis with effusion does not stop here, but an exudate of serum containing desquamated and proliferated endothelial cells, scanty pus-corpuscles, and flocculi of fibrin is poured out to an amount varying from 200 cubic centimeters to 2 liters. The serum may be admixed with blood in old and cachectic patients. This effusion fills the pericardial sac, displaces the heart upward and to the left, and may be abundant enough to exert pressure on the heart, especially the auricles and the venæ cavæ, the left bronchus, the œsophagus, and the lung, or to exert traction on the recurrent laryngeal nerve.

In mild cases the myocardium is turbid and pale to the depth of from 2 to 3 millimeters, but in more severe cases the myocarditis is more extensive and may endanger the life of the patient. Pericarditis is often associated with endocarditis, as both diseases are apt to arise from the same causes. In rare cases inflammation may spread from the one to the other membrane.

Pericarditis may terminate in the following ways: (1) By resolution and absorption of both fibrinous and serous exudate. (2) By organization of the fibrin into connective-tissue adhesions. From such an adherent pericardium may result hypertrophy and dilatation of the heart, often to an extreme degree, chronic interstitial myocarditis, and fatty degeneration of the heart. (3) The inflammation may become chronic. The pericardium becomes thickened by connective-tissue growth and may be infiltrated with salts of lime, and the fluid may not be absorbed. This condition is especially seen, however, in tubercular cases.

The **symptoms** may be divided into four groups:

1. *Inflammatory Symptoms.*—An initial chill is rare. Fever is not usually high; it may be irregular or absent. Hyperpyrexia has been observed in rheumatic cases. Pain, which is noted in about three-quarters of all the cases, may be distressing and intense, resembling angina, or the patient may complain only of a feeling of uneasiness, may be, or oppression. The pain may be referred to the precordia or to the lower part of the sternum. The pain usually diminishes as fluid accumulates, but in some cases it becomes thereby increased. It may be increased when the patient inclines forward or when pressure is exerted upward from the epigastrium. There may be tenderness on pressure over the precordia. These inflammatory symptoms may be slight, the onset of the disease being insidious. There may be only a gradual failing in health, slight dyspnœa, and increasing pallor.

2. *Deranged action of the heart* is seen in both forms of pericarditis, and depends upon the associated myocarditis. If there be no myocarditis, the pulse may be rapid and irregular or strong and tumultuous. In dry pericarditis of a small area the pulse may be but slightly altered. If myocarditis be extensive, the power of the heart is thereby weakened and symptoms of heart failure ensue. The pulse becomes rapid, feeble, and irregular; the patient feels faint and complains of dyspnœa. In bad cases there may even be venous congestions, especially if the venæ cavæ be pressed upon by the distended pericardial sac. The presence of venous congestions is almost a proof of the presence of myocarditis or complicating endocarditis.

3. *Pressure-symptoms* occur with abundant effusion. The heart's action becomes irregular and weak; the veins of the neck and the arm may become congested and enlarged. Dyspnœa may be aggravated by pressure on the left bronchus. Pressure or traction on the recurrent laryngeal nerve as it winds around the aorta will produce hoarseness or aphonia, laryngeal cough, and dyspnœa which may be spasmodic and distressing. With large pericardial effusions may be observed the *pulsus paradoxus*, in which the pulse-wave is weak or even imperceptible during inspiration.

4. *Symptoms of the primary disease* often coexist with those of the pericarditis, and may obscure the diagnosis. To be on the safe side, the temperature and the pulse should be recorded in every disease that may cause pericarditis, especially rheumatism and scarlet fever, even into convalescence, and physical examinations of the heart should be made regularly. There should be special inquiry as to pain and dyspnœa. Pericarditis should be suspected in every case of Bright's disease seriously sick with obscure symptoms.

Physical Signs.—Palpation may reveal a friction fremitus best marked over the right ventricle. The characteristic sign is the friction sound. It is of a rubbing, grating, or sticky quality, and may in some cases resemble the creaking of new leather. It may occur during the systole, during the diastole, or during both, the last being the most common. In rare instances it has a triple rhythm. It is not exactly synchronous with the heart sounds, the double or to-and-fro movement usually outlasting the first and second sounds. The friction sound appears superficial close to the ear and is intensified by pressure with the stethoscope or by inclining the patient forward. It is heard best over the exposed portions of the heart—that is, in the fourth and fifth spaces near the sternum. It may, however, be heard at the extreme base or apex. It is not transmitted in any direction, as are endocardial murmurs. The friction sound is inconstant, coming and going without apparent cause, and it suffers frequent changes in character and point of maximum intensity.

In cases with effusion the friction râle may disappear as the pericardial surfaces are separated, or the râle may be heard at the upper part of the heart. It reappears with the absorption of the fluid. On inspection there may be bulging of the precordia, especially in children. The antero-lateral region of the chest may even be enlarged. Bulging of the precordia without visible pulsation is distinctive.

Palpation may detect fluctuation in rare instances. The apex beat is usually displaced upward and outward, and the cardiac shock is often effaced. It is distinctive to have the apex beat well within the limits of cardiac dulness. The strength and location of the apex beat are largely influenced by the position of the patient.

Percussion shows an increased area of cardiac dulness of a pyramidal shape, the apex being up, the broad base of the dull area producing a flat note in the fifth and even in the sixth right interspace. The dulness has an abrupt outline and extends beyond and below the apex beat. The dulness extends above the normal limits, so that very marked dulness above the third rib should

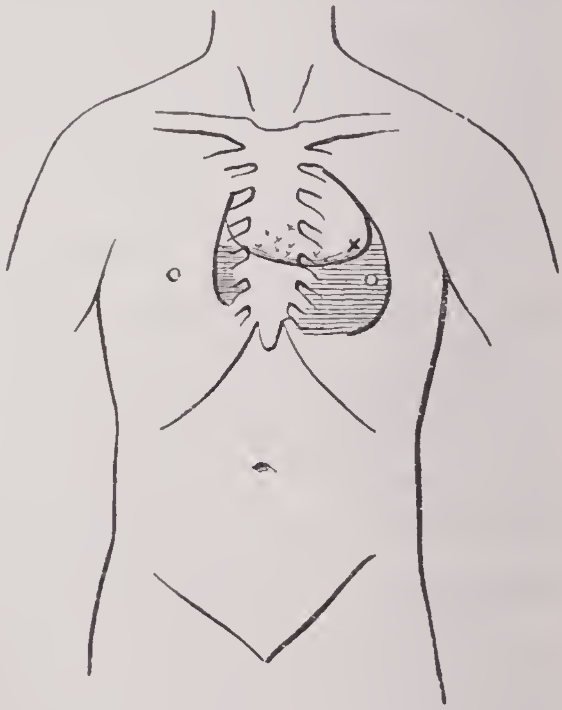


Fig. 5.—Pericarditis with effusion, showing the area of dulness, the position of the heart within the pericardial sac, and the location of the apex beat (marked ×). Small crosses mark the position of the pericardial râles.

lead to the suspicion of effusion. The surrounding border of the lung frequently yields a tympanitic percussion note from its relaxation. An abundant effusion extending to the left may be mistaken for pleurisy. With the patient in a sitting position, dulness, increased vocal fremitus, and bronchial voice and breathing may be detected over a small area under the angle of the left scapula. These signs disappear when the patient leans forward or assumes the knee-chest position; they are due to the pressure of an abundant effusion upon the lower lobe of the left lung, and they afford valuable aid to diagnosis, especially in children and young adults.

On auscultation the friction râle may be heard at the base of the heart; the sound is inconstant, coming and going. It returns with the absorption of the fluid. The heart sounds are feeble and may become inaudible.

Clinical Types.—1. *Latent Cases.*—In these cases the symptoms are obscured and a diagnosis is not made during the life of the patient. Such cases commonly occur with advanced Bright's disease.

2. *Mild Cases.*—In these cases there is no appreciable amount of effusion and the myocardium is not involved. These cases give symptoms of inflammation and of slight derangement of the heart's action.

3. *Severe cases* are those with abundant effusion associated with myocarditis. Symptoms of inflammation and cardiac failure occur, and pressure-symptoms appear according to the amount of the effusion.

4. If complicated by previous or coexisting endocarditis, compensation may be upset and symptoms of cardiac failure with venous congestions may occur. The presence of these latter symptoms indicates more than mere pericarditis: they point always to a complicating myocarditis or endocarditis.

5. In some cases delirium, mania, or hallucinations may give to the disease a cerebral character.

The **diagnosis** in many cases is exceedingly obscure. The points of diagnosis from dilated heart are concisely given by Sansom:

	Pericarditis.	*Dilatation.*
Outline of dulness.	Dulness pear-shaped, and the enlargement chiefly upward.	Dulness not pear-shaped, and enlargement chiefly downward.
Rate of development of dulness.	Often rapid and then characteristic.	Usually very slow, though a rapid dilatation of the heart sometimes occurs.
Impulse and apex beat.	The impulse when present is in the third or fourth left interspace; apex beat tilted upward and outward or effaced.	Impulse can usually be felt to the left of the lower end of the sternum or in the epigastrium.
Relation of dulness to left apex beat.	Dulness may extend to the left of the left apex beat.	Dulness does not extend to the left of the left apex beat.
Pain over precordium and tenderness in epigastrium.	Often present.	Usually absent.
Pulsation in veins of the neck.	May be present if endocarditis complicates.	Often present when right heart is dilated.
Etiology.	Usually acute, in course of acute rheumatism, cirrhotic Bright's disease, etc.	Usually chronic; often associated with chronic valvular lesions, fatty and fibroid degeneration, anæmia, etc.
Fever.	Often present.	Absent unless from some complication.

The characteristics of the friction râle already described should prevent its being mistaken for endocardial murmur.

Pleuritic frictions may be so modified by the movements of the neighboring heart as to resemble closely the pericardial rub. The pericardial friction is less liable to variation and is best heard when the heart is least covered by lung—that is, during expiration—while with the pleuro-pericardial sound the reverse is true.

Pericardial effusion will not be mistaken for pleurisy with effusion if careful examination be made.

The **prognosis** generally is good. It depends (1) upon the cause of the disease, being worse in distinctly septic cases and in old people with advanced nephritis; (2) upon the severity of the lesion; and (3) upon the condition of the heart itself—how it does its work, and whether it be the seat of myocarditis or of previous or complicating endocarditis. The prognosis of cases occurring with pneumonia is better than that of any other form.

Treatment.—The patient should be put to bed and kept absolutely quiet mentally and bodily. The bowels should be opened by calomel and Epsom salt. To control the inflammation continuous application of cold by ice-bags or by Leiter's tubes is often of the greatest service. Counter-irritation by cups, poultices, blisters, or tincture of iodine may be resorted to, and local blood-letting by leeches applied to the precordium may be employed in robust sthenic cases. Small doses of opium are serviceable in steadying the heart and controlling the symptoms. Aconite should be given in case of tumultuous heart-action, to reduce the work of the heart to a minimum.

Should symptoms of a failing heart appear, stimulants are indicated, whiskey and digitalis being the most efficient.

To promote absorption of the effusion, blisters over the precordium are recommended. The bowels should be opened freely, if the patient is not debilitated by over-catharsis, and diuretics should be given, an efficient combination being the infusion of digitalis with iodide and acetate of potassium.

When the effusion is giving rise to pressure-symptoms and cannot be absorbed by the foregoing measures, aspiration may be performed. Puncture is best made in the fifth left interspace about two inches from the sternum—roughly, therefore, in the position of the normal apex beat.

PURULENT PERICARDITIS.

Etiology.—This form of pericarditis, which results from infection by the bacteria of suppuration, complicates the septic and pyæmic diseases already alluded to as causative of pericarditis. The disease is frequently seen with influenza. Infection of a serous effusion may occur from the use of an infected aspirating needle. It may occur with pneumonia due to diplococcus infection. The tubercular form of purulent pericarditis will be described in a separate article.

Pathology.—The pericardium is thickened and is infiltrated with fibrin, serum, and pus-cells; its superficial layers, which are converted to granulation-tissue, may present distinct ulcerations. The pericardium is usually covered with a thick layer of fibrin and pus. The effusion is distinctly purulent and is usually abundant, at times exceeding three liters. The myocardium is always more or less involved; it may be the seat of an acute myocarditis or of a fatty degeneration.

The suppurative process may extend in any direction, so that the effusion may rupture into a bronchus or into the trachea, or may appear externally through the thoracic wall. In rare cases the pus may be absorbed partially, cheesy deposits alone remaining on the surface of the pericardium, which becomes thickened and may be infiltrated with lime salts. Should the pus be withdrawn by operation, the pericardium may become thickened and adherent, or it may remain as a chronic suppurating surface discharging its contents through a thoracic fistula.

In long-continued cases amyloid changes occur in the arteries, spleen, liver, and kidneys, and chronic diffuse nephritis is likely to develop.

The **symptoms** are generally those of serous pericarditis. The inflammatory symptoms are the same, but they are likely to be obscured by those of the original septic disease, so that the pericarditis may escape unnoticed. The symptoms of mechanical pressure are identical with those of the serous form. Myocarditis, however, being more extensive, gives prominent symptoms—heart failure, rapid and feeble pulse, cerebral anæmia, dyspnœa, and even venous congestions. Septic symptoms make their appearance and indicate the presence of pus—erratic chills followed by sudden rises in temperature, cold sweating, rapid and feeble pulse, diarrhœa, and a low form of delirium.

The **physical signs** are those of pericardial effusion. Should pointing occur, the skin becomes red and shining and fluctuation may be detected. In case of doubt as to the nature of the effusion, hypodermic aspiration under antiseptic precautions should be employed.

The **prognosis** is bad. The primary septic disease may of itself be fatal. The myocarditis is apt to lead to dangerous heart failure, and even if the effusion be removed by operation, the pericardium never returns to a normal condition, and the danger of adhesive pericarditis should be considered. The prognosis of cases complicating pneumonia is less grave than in the other forms.

Treatment is entirely surgical. The pericardium should be opened freely and drained. The opening should usually be made in the fifth interspace, one and a half inches from the edge of the sternum.

CHRONIC ADHESIVE PERICARDITIS.

Etiology and Pathology.—Thickening of the pericardium and the formation of connective-tissue adhesions between its opposed surfaces follow dry pleurisy or the absorption of the serous effusion. This condition may also follow absorption of a sero-purulent exudate, the pericardium being thickened by connective tissue covered with a cheesy deposit and often infiltrated with salts of lime. If the adhesions are such as not to impede the action of the heart, there may be little or no change in its structure. If the adhesions are firm and short, the impediment afforded to the free action of the heart leads to hypertrophy, frequently of enormous dimensions. Thus compensation is established. Fatty degeneration and dilatation finally result in heart failure. Pleuro-pericardial adhesions are usually present.

Symptoms.—As long as the heart's power is good, symptoms are indefinite and may be absent. When myocardial degeneration and dilatation ensue, symptoms of failure of cardiac power result, as shown by arterial anæmia and venous congestions. The pulse becomes weak and irregular, and the *pulsus paradoxus* may be noted. There may be attacks of angina, and sudden death may occur. There are frequently observed recurring attacks of acute dry pericarditis.

Physical Signs.—The heart is usually hypertrophied. It may be dilated, and there may be the systolic apical murmur of relative incompetence of the mitral valve. A significant sign is the retraction of the apex during systole. This sign, however, is not absolutely diagnostic, and it may be absent. After the systolic retraction the chest-wall may suddenly rebound during diastole, often with a sharp shock. The rapid rebounding of the chest-wall may suddenly empty the jugular veins, giving rise to the "diastolic collapse," or Friedreich's sign.

The heart sounds may be normal, or muffled if the pericardial layers be greatly thickened. There may be distinct and persistent friction sounds, often of a grating, creaking, or "new-leather" character.

The **prognosis** is not good. Compensation is almost certain to be upset by myocardial degeneration and dilatation. Sudden death often occurs.

Treatment consists in steady counter-irritation applied to the precordium, and the treatment of cardiac weakness when it occurs. Avoidance of severe or sudden exertion is to be enforced strictly.

TUBERCULAR PERICARDITIS.

Tuberculosis of the pericardium may be part of a general infection or it may be a local disease. It is of rare occurrence, and usually results from extension from tubercular disease of the bronchial or mediastinal glands, the lungs, or the pleuræ, although in many cases the primary lesion may be so slight as to pass unnoticed.

Pathology.—Four forms of the lesion are seen: (1) The pericardium is thickened by imbedded tubercules; its surface is congested and covered with fibrin. (2) The pericardium resembles the first form, but there is an exudate of serum as well. (3) The pericardium is bright red and studded with white points, which are the tubercules. There is a hemorrhagic effusion. (4) The pericardium is thickened and infiltrated with connective-tissue cells and masses of tubercle-tissue; its surface is coated with fibrin, pus, and broken-down tubercle-tissue. There is a purulent effusion often containing shreds of cheesy tissue in which the bacilli are to be found.

The **symptoms** of tubercular pericarditis resemble those of the non-tubercular forms in essential features and physical signs. They differ in the following particulars: (1) The disease is insidious, the first symptoms usually being weakness, loss of flesh, and anæmia. (2) The symptoms are persistent, without tendency to improvement. The effusion recurs in spite of drug-treatment or aspiration. (3) The course of the disease is steadily downward. Recovery is possible, though rare, and is not to be expected except in slight lesions.

Diagnosis is aided by the absence of the ordinary causes of pericarditis and by the presence of tubercular lesions, especially within the thorax. Double pleurisy with effusion or with pericarditis is almost always tubercular. Hemorrhagic effusion may occur in pericarditis of the aged or the scorbutic, but is much more significant of tuberculosis or of cancer. The finding of tubercle bacilli in the pericardial effusion will of course settle the diagnosis past all dispute.

Treatment is that of the pericarditis after established prinples, and of the general condition by the supporting treatment that is employed in all tubercular cases.

CANCEROUS PERICARDITIS.

Cancer or sarcoma of the pericardium is rare and is almost never primary. Besides the malignant new growths the pericardium presents the lesions of simple inflammation with serous, or more frequently with hemorrhagic, exudate.

The **symptoms** are those of a slowly-developing pericarditis with effusion. Pain is often lancinating and excessive. Aid is afforded in diagnosis by the hemorrhagic character of the effusion (as in tubercular peritonitis), the presence of the primary malignant growth, and the symptoms of cancerous or sarcomatous cachexia.

The **prognosis** is invariably bad.

Treatment is merely palliative. Injections of toxic products of erysipelas germs may be employed in sarcomatous cases.

HYDROPERICARDIUM.

Large serous effusions without inflammatory signs, constituting "dropsy of the pericardium," occur in connection with general dropsy, usually due to kidney or heart disease.

Symptoms.—Associated with dropsical effusions in the pleural cavities, this condition embarrasses the action of the heart and the lungs. The disease may occur after scarlet fever without general dropsy. There are no inflammatory symptoms.

Physical Signs.—The physical signs are those of effusion, but there is no friction râle.

HÆMOPERICARDIUM.

Blood in the pericardium results from rupture of an aneurysm of the first part of the aorta, of the coronary arteries, or of the heart itself, from rupture of the heart-wall in myocarditis, or from penetrating wounds or severe crushing of the thorax.

The **symptoms** are those of heart failure and of hemorrhage in general. When the hemorrhage occurs slowly, the symptoms of heart failure may be obscured by nausea and vomiting. Pericarditis with bloody effusion cannot properly be called hæmopericardium.

The **prognosis** is bad. Recovery, however, may occur in case but little blood has been lost.

PNEUMOPERICARDIUM.

Air in the pericardial sac may occur from penetrating wounds of the chest or from perforation of the lungs, œsophagus, or stomach. Gas may result from the decomposition of a purulent exudate. Acute pericarditis is always excited, in most cases with a purulent exudation.

The **symptoms** are those of cardiac failure—fever and pericardial effusion. Death usually follows rapidly.

Physical Signs.—It is characteristic of pneumopericardium to have a movable area of dulness over the fluid, with an overlying area of tympany over the gas. The heart sounds may be distant and feeble but of a metallic gravity. There are splashing, churning sounds of a metallic quality with frictions.

The **diagnosis** is to be made from dilated stomach, sacculated pyopneumothorax, large superficial pulmonary cavity, and in rare cases from hernia of intestine through a congenital opening in the diaphragm.

The **prognosis** is almost hopeless except in recent traumatic cases.

Treatment consists in free incision and drainage.

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2. DISEASES OF THE HEART.

HYPERTROPHY AND DILATATION.

The heart is an organ with a definite function—to maintain the circulation of the blood—and its diseases are important only to the extent to which they modify this function. *The effect of all cardiac lesions is one: the heart is prevented from doing its work.* The variety of the lesion is less important than its results on the power of the heart.

There are three things to be considered in the work of a normal heart: (1) The heart is a hollow muscle contracting on its contents; (2) the heart drives the blood against resistance; (3) the onward flow of blood is facilitated by the perfect condition and action of the valves. Any one of these three conditions may become deranged, and so cause interference with the work of the heart.

Causes Preventing the Heart from doing its Work.—I. Weakness of the Heart-muscle.—This weakness may be either comparative or actual.

1. *Comparative Weakness.*—Here the heart, though normal in strength, is not strong enough to do an extra amount of work that may be required of it. This condition is seen in severe and sudden *over-exertions*, as in rowing-races, especially without previous training, or in exertion, even to a moderate extent, in high altitudes, or

when the proper expansion of the chest is prevented, as by the heavy cross-straps of soldiers on the march, the action of the heart becoming secondarily embarrassed and insufficient to meet the demands made upon the organ.

2. *Actual weakness* is the more important form, not only because it is continuous in its action, but also because the heart is rendered unfit to meet the demands of ordinary life. The heart-muscle may become weakened in the following ways:

(*a*) *By degeneration*, either acute, as in fevers and infectious diseases, or chronic, as in fatty heart.

(*b*) *By inflammation*, either acute, as in acute myocarditis, which so commonly complicates pericarditis, or chronic, as in chronic interstitial myocarditis.

(*c*) *By poor nutrition.* (1) In some cases the nutrition of the whole body suffers as well as the heart, as during convalescence from a prolonged fever or in the case of profound anæmia, the heart as well as the other organs being poorly supplied with blood, frequently giving rise to dilatation and cardiac symptoms. (2) In other cases the heart alone suffers. This condition arises when, by reason of atheroma of the coronary arteries, the supply of blood to the heart-muscle itself is lessened.

II. Increased Peripheral Resistance.—It is important to remember that increased peripheral resistance may occur either in the systemic vessels, interfering in consequence with the action of the left side of the heart, or in the pulmonary vessels, interfering with the work of the right heart. Each system, then, must be considered separately.

1. *Increased peripheral resistance in the systemic circulation, affecting the work of the left heart.*

(*a*) *By atheroma or aneurysm of the aorta* or great vessels.

(*b*) *By endarteritis*, because of the narrowed lumen and the non-yielding walls of the small arteries.

(*c*) *By spasm of the small arteries.* The spasm may occur as (1) the result of an endarteritis, or (2) from Bright's disease, (3) uric-acid diathesis, (4) gout, (5) diabetes, or (6) over-eating and excessive drinking.

2. *Increased peripheral resistance to the pulmonary circulation, affecting the work of the right heart.*

(*a*) *By failure of the left heart,* either from its muscular weakness, from dilatation, or from valvular disease, especially lesions of the mitral valve.

(*b*) *By obstruction of the pulmonary vessels* by spasm, endarteritis, or obliteration. This condition occurs in emphysema, in interstitial pneumonia, and to some extent in pulmonary phthisis.

III. ERROR IN THE VALVES.—1. *By stenosis* of any valve, the valve-orifice becoming narrowed, there is difficulty in the onward passage of the blood.

2. *By insufficiency* of any valve, so that blood can regurgitate backward instead of being prevented from so doing by perfect closure of the valve.

Valvular insufficiency may result in one of four ways:

(*a*) *By inflammation,* causing thickening and contraction of the valve.

(*b*) *By rupture* from violence or from ulceration.

(*c*) *By stretching of the orifice* to which the valve-segments are attached. This applies especially to the mitral valve. The mitral-valve segments can close the left auriculo-ventricular orifice, provided it be of normal size; but should the left ventricle become dilated, the auriculo-ventricular orifice becomes stretched to too great a size to be covered by the valve-segments during their closure, hence regurgitation of blood is permitted at the time of the ventricular systole. This condition is often spoken of as "relative incompetence."

Regurgitation into the left ventricle may occur at the time of the ventricular diastole, past the aortic valve if the aortic ring be stretched by an aneurysm of the aorta near the valves, even if the latter be normal.

(*d*) *By poor ventricular contraction.* The relation of muscular fibres to the mitral ring is such that when the fibres contract the auriculo-ventricular orifice is reduced to one-half its size and can then be covered by the normal valve-segments. If the contraction be poor, the orifice is not so much diminished in size, and cannot be closed entirely by the valve-segments. In this way regurgitation of blood past the mitral valve is permitted at the time of ventricular contraction. To this condition the name of "relative incompetence" is also applied.

Methods of Compensation.—When an extra amount of work is required of the heart in any of the above-mentioned ways, the great and important question is whether the heart can rise to the emergency and meet the demand for the extra amount of work. When the increased power of the heart becomes equal to the increased demand, then the circulation is again in an equilibrium and "compensation" is established. Upon the question of compensation the whole prognosis and treatment of cardiac diseases depend. Compensation occurs in two ways:

1. *By increase of the force and frequency of the heart's action.* This is the simplest form of compensation, and is especially adapted to meet sudden demands. The rapid and forcible heart's action after a short run is the best example that can be cited. Compensation by increase of the heart's action is often associated with hypertrophy, and it is most important that this fact be remembered. For example, in aortic regurgitation hypertrophy of the left ventricle is hardly efficient enough by itself to compensate for the diastolic regurgitation of blood. There must also be an increased frequency of ventricular contraction to keep the ventricle

emptied of the blood which, during diastole, pours into it not only from the auricle, but backward as well from the aorta, past the inefficient aortic valve. It is poor practice to try to reduce the pulse-rate of such a case to the normal limit of 72 to the minute. If we do, we run the risk of giving the ventricle time enough between contractions to become distended to too great a limit, with disastrous results.

2. *By hypertrophy.*

HYPERTROPHY.

Hypertrophy is an actual increase in the amount of cardiac muscle, giving the heart thereby increased force adequate to the increased demand for force. The condition is purely compensatory and physiological, and is analogous to the enlargement of the biceps of a blacksmith. Certain conditions must be complied with before hypertrophy can occur.

1. *A certain amount of time* is necessary. Hypertrophy is a slow process, requiring a minimum time of two weeks for its development. Preceding its completion there may be a primary compensation by increased force and frequency of the heart's action. The process of hypertrophy is so slow that patients may die from disturbed circulation before compensation is established.

2. *The lesion must not be excessive.* It can readily be seen that in very extensive lesions no amount of muscular hypertrophy can restore the circulation to an equilibrium.

3. *The lesion must not be too rapidly progressive.* A lesion slight at first may be perfectly compensated, but if it progresses it may become too severe for hypertrophy to keep up with it.

4. *There must be a healthy condition of the heart-muscle.* This is of the utmost importance. Weakness of the heart-muscle may prevent hypertrophy entirely or may only allow

a degree of hypertrophy inadequate to the demand, so that compensation occurs but imperfectly. Weakness of the heart-muscle may at any time prevent the maintenance of hypertrophy, so that compensation will fail. The treatment of a hypertrophied heart consists solely in the maintenance of a proper amount of hypertrophy, and this state depends in the greatest degree upon the condition of the heart-muscle itself. It is important, then, to consider in what ways the heart-muscle may be weakened.

Local Causes.—(1) *Degeneration*, either fatty, fibroid, or the acute degeneration of fevers; (2) *inflammation* as complicating acute endocarditis or pericarditis; (3) *poor blood-supply*, from atheroma of the coronary artery or from severe general anæmia.

General Causes.—(1) General weakness and debility; (2) old age; (3) anæmia; (4) lack of proper care of self; indulgence in alcohol and tobacco, irregular hours and habits, exhausting work, improper food, etc.

Symptoms of Hypertrophy.—If there be compensatory hypertrophy adequate to meet the extra demand on the heart, the circulation will be *in perfect equilibrium and there will be no subjective symptoms.* The diagnosis is made by the finding of some cause demanding the performance of extra work. This cause may be valvular error or increased peripheral resistance from endarteritis or from the arterial spasm of Bright's disease, or from any of the other mentioned causes, but in all cases alike perfect compensation allows of no circulatory disturbances.

Physical Signs of Hypertrophy of the Left Side of the Heart.—*Inspection* may show some bulging of the precordia, especially in children, in whom asymmetry of the chest may become apparent. The area of visible impulse is much increased, and the apex beat is seen to be displaced downward and outward.

Palpation.—There occurs over the lower part of the heart a slow, heaving impulse which is one of the most distinctive signs of simple hypertrophy. The apex beat occurs as a powerful, deliberate thrust, and is felt downward and outward from its normal situation. It may be felt in the sixth, seventh, or eighth intercostal space and outside the nipple line. The position of the apex beat is a good guide to the position of the left border as found by auscultatory percussion. If the apex beat be continuously strong and heaving, it proves a healthy condition of the myocardium and is of good prognostic value. The character of the apex beat aids in differentiating hypertrophy from dilatation. In some patients with deep, well-covered chests, and in those whose lungs are emphysematous, overlapping the heart to an abnormal degree, the signs by inspection and palpation may not be apparent.

Percussion should by preference be performed by the auscultatory method, as greater accuracy is thereby assured. There is increase in the transverse area of dulness, so that the left border lies distinctly outside the nipple line. A transverse diameter of over four and a half inches is never seen in a normal heart. In some cases the transverse diameter is not increased by percussion, so that the diagnosis must be made by other signs. There is also an increase in the vertical direction, the upper limit of cardiac dulness being frequently in the second intercostal space.

Auscultation.—In hypertrophy without valvular disease the sounds of the heart may be normal, or the first sound at the apex may be prolonged and of a booming, muscular quality. Reduplication is common in the hypertrophy consequent upon kidney disease. The second sound heard over the aortic area is loud, clear, and snappy, being "accentuated." This accentuation of the second aortic sound is heard best in hypertrophy consequent upon increased arterial resistance. For its development there must be not only increased resistance in front, but also increased force from behind, so that at each systole blood is pumped into the aorta with force enough to give rise to tension high enough to overcome the increased peripheral resistance. The accentuation is due to the forcible closure of the aortic valves at the beginning of diastole by reason of this high arterial pressure. The very presence of an accentuation of the second aortic sound is of good prognostic significance, as it proves necessarily that the heart is able to do its extra work. The disappearance of the accentuation and the weakening of the second aortic sound imply weakening of the heart's power, and are of bad import.

In hypertrophy dependent upon valvular lesions the heart sounds are necessarily altered and are replaced or associated with murmurs.

In hypertrophy associated with dilatation the physical signs are correspondingly modified.

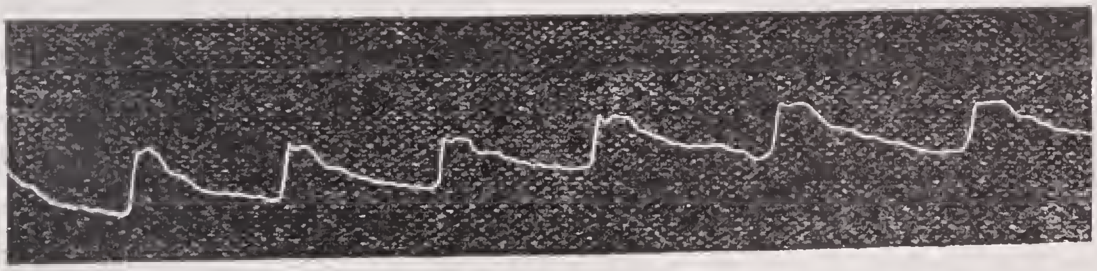


FIG. 6.—Sphygmogram showing high tension in Bright's hypertrophy.

The pulse of hypertrophy not due to valvular disease is full, strong, and of high tension. Its characteristics are best seen in cases of hypertrophy consequent upon increased peripheral resistance, as endarteritis in Bright's disease.

Physical Signs of Hypertrophy of the Right Side of the Heart.—*Inspection* may reveal bulging of the lower part of the sternum. Below the ensiform cartilage there is a visible pulsation due to the enlargement downward of the hypertrophied right ventricle.

Palpation.—There is a distinct heaving impulse appreciable just below the ensiform cartilage. This epigastric pulsation is seen and felt also in displacement of the heart downward from aneurysm, from mediastinal tumors, and from emphysema, so that the diagnosis of hypertrophy of the right ventricle should always be corroborated by other signs. The second sound heard over the pulmonary area (second space to the left of the sternum) is accentuated on account of increased tension within the pulmonary artery. This sign is of good prognostic value. The diagnosis is aided by the finding of the cause for the hypertrophy either in a failing left ventricle or in some condition causing increased resistance in the pulmonary circulation.

DILATATION.

Dilatation is reached whenever the ventricle does not empty itself during the systole. There are two principal causes of dilatation:

1. *Increased blood-pressure within the cardiac cavities,* from increased peripheral resistance, from valvular disease, or from excessive muscular effort. An extra amount of work is thrown on the heart, which fails to respond by hypertrophy, either by reason of a too sudden, too extensive, or too rapidly progressive lesion, or by reason of impaired vitality of the cardiac muscle.

2. *Impaired nutrition of the heart-walls* may so weaken their resisting power that dilatation occurs even if the heart be not called upon for extra work. The condition is often spoken of as "idiopathic dilatation." The result in either case is the same: *the heart can no longer do its work.* Three conditions necessarily result: (1) The arteries are underfilled with blood—arterial anæmia. (2) The veins are overfilled,

and there are venous congestions of the various viscera that may be acute or may become chronic. (3) The heart cannot empty itself completely at each systole, either from too much work required or from deficient contraction-force, so that there is always a certain amount of residual blood which increases the size of the cardiac cavities and *dilates* them, especially if the cardiac muscle be weak and degenerate.

Hypertrophy is a compensatory process; dilatation is destructive. Hypertrophy is an evidence of vigor; dilatation is an evidence of weakness. In hypertrophy the work of the heart is well done; in dilatation the work is imperfectly performed.

Dilatation may occur by itself or may be associated with hypertrophy. In some cases hypertrophy may precede, compensating for some pre-existing lesion; then compensation fails and dilatation gradually ensues. In other cases, from a sudden lesion the heart is at first dilated, compensatory hypertrophy occurring after a time.

Symptoms.—If the left ventricle dilates, its walls become weakened and thinned and the mitral ring becomes stretched. From both of these reasons, as previously explained, there is apt to be developed "relative incompetence of the mitral valve," the blood being regurgitated at the time of the ventricular systole into the auricle, and so exerting a back pressure and congestion in the pulmonary vessels. If the right ventricle be strong and of good nutrition, it will act forcibly and will hypertrophy, forcing the blood well into the pulmonary blood-vessels against this backward pressure, standing thus as a barrier between the dilated left ventricle and the systemic venous system. If, on the contrary, the right ventricle be not strong enough to overcome the backward pulmonary pressure and to sustain the circulation of blood in the lungs, the ventricle will dilate, its contraction-force will be weakened, there will be back pressure of blood in the veins of the body, and general venous congestion of the viscera will ensue. The consideration, therefore, of the symptoms of dilatation is largely that of venous congestions.

ACUTE VENOUS CONGESTIONS.—*Brain.*—The pia mater is congested and œdematous. There is an increase in the cerebro-spinal fluid. Clinically there are developed delirium with delusions, insomnia, stupor, and headaches.

Lungs.—Congestion and œdema exist, especially marked posteriorly at both bases. There may be areas of hypostatic pneumonia. Clinically there is developed dyspnœa, at first only on exertion, later becoming steady and of the variety known as "orthopnœa." There is a cough with expectoration which may contain blood. The expectoration may be serous and profuse. On physical examination are found moist bronchial râles and crepitations, either general or at the bases alone, and there may be the physical signs of consolidation in case of hypostatic pneumonia.

Pleura.—There is hydrothorax. This gives rise to symptoms due to the mechanical presence of the fluid—dyspnœa, cough, and the displacement of neighboring viscera. There are the physical signs of fluid in both pleural cavities.

Stomach.—There is congestion of the gastric mucosa, giving rise to vomiting, to occasional small hemorrhages, and to symptoms of gastric indigestion.

Intestines.—There is either diarrhœa or constipation and a decided loss of general nutrition.

Liver.—There are congestion, an enlargement which is slight and symmetrical, symptoms of disturbances in the functions of the liver, and frequently slight jaundice.

Spleen.—There is moderate enlargement.

Peritoneum.—There is a serous peritoneal effusion known as "ascites," with enlargement of the abdomen; mechanical symptoms of the effusion and the physical signs of its presence also exist.

Kidneys.—The kidneys are enlarged and congested. The urine is diminished; albumin and casts are present in moderate amounts. A practical rule is that whenever the urine is turbid and deposits urates day after day irrespective of diet, mode of life, or exercise, a failing heart should be looked for.

Skin.—Cyanosis appears; there is also œdema, at first appearing in the feet and ankles after standing, but later becoming general.

CHRONIC VENOUS CONGESTIONS.—*Brain.*—The pia mater is congested and œdematous, and the fluid in the ventricles is increased—the so-called "wet brain." There is apt to be considerable atrophy of the cerebral cortex. Clinically there are headache, attacks of dizziness, black specks before the eyes, buzzing noises in the ears, insomnia or unnatural stupor, and delirium with delusions.

Lungs.—There is a chronic bronchitis with cough and expectoration. There may be repeated small hemorrhages. Dyspnœa is present at first on exertion, but later becomes steady and of the orthopnœic variety. In chronic cases there is developed the chronic congestion of the lung known as the "pneumonia of heart disease," or "pigment" or "brown induration." The lung is dry and leathery and is mottled brown and salmon pink in color. Microscopic examination shows dilatation and lengthening of the capillaries in the alveolar wall, so that by their loopings they encroach upon the air-spaces. The alveolar wall is thickened by the growth of its capillaries and by that of muscular fibres and new connective tissue. In the walls of the alveoli and in their endothelial cells there is a deposit of pigment due to small punctate hemorrhages from the congested and tortuous capillaries. The endothelial cells are increased in number, in some places so filling the cavity of the alveoli as to form patches of hepatization.

Pleura.—There is fluid in both pleural cavities, giving rise to mechanical symptoms and physical signs.

Stomach and Intestines.—There is either chronic congestion or a chronic catarrhal inflammation, giving rise to characteristic symptoms.

Liver.—There is the so-called "nutmeg liver," its name arising not from any surface irregularity, but from the mottled color. The liver may be large or small or normal in size. The centre of each acinus is pigmented and depressed; the periphery is yellowish from fatty degeneration of the liver-cells. There may be a catarrhal inflammation of the bile-ducts with jaundice; there may also be an associated cirrhosis.

Peritoneum.—There is ascites.

Kidneys.—There may be either chronic congestion or chronic diffuse nephritis. In the former case the urine is diminished, of increased specific gravity, and may contain a little albumin. The quantity of urea is normal. There are no characteristic clinical symptoms. In the case of chronic diffuse nephritis the urine, which may be either diminished or increased in quantity, contains a diminished amount of urea. There may or may not be albumin and casts. The specific gravity of the urine is regularly low, being about 1010. This form of nephritis is apt to give symptoms of chronic uræmia.

The occurrence of nephritis with cardiac dilatation is not always the same.

1. In some cases the dilatation begins first from valvular disease or from any other cause giving rise to venous congestions, including congestion of the kidney, which develops into chronic diffuse nephritis.

2. In some cases there is first developed a chronic diffuse nephritis, by reason of which toxic products, not being properly excreted, collect in the blood, causing endarteritis and arterial spasms. This condition increases peripheral resistance, which is met by hypertrophy of the left ventricle. Should this compensation fail, there will be added dilatation of the heart.

3. In other cases hypertrophy with subsequent dilatation and chronic diffuse nephritis occurs as part of the disease process known as "arterio-capillary fibrosis."

Skin.—There may be cyanosis. There is a characteristic obliteration of the fine lines and wrinkles of the skin of the face, due to its congestion and moderate œdema. There is œdema, first noticed in the ankles on walking or standing, later becoming more generally distributed.

Physical Signs of Dilatation.—1. *Of Dilatation of the Left Heart.*—*Inspection* shows a diffuse undulatory pulsation over a large area. There may be, however, no pulsation visible.

Palpation.—The impulse is vibratory and diffused. There are cases in which the pulsation can be seen but cannot be felt. The apex beat is poorly defined; it is diffused, weak, snappy, or absent.

Percussion.—There is enlargement of cardiac dulness both vertically and transversely. The left border of the area of dulness is frequently found as far as the anterior axillary line. The upper border may be at the second rib; the apex may be carried downward as far as the seventh or eighth rib. The increase of size is in the same direction as in hypertrophy, but is carried to a greater extent.

Auscultation.—The first sound at the apex is short and snappy, approaching the character of the more purely valvular second sound. The second sound at the base is weak or absent. Both of these signs are important in making a diagnosis between dilatation and hypertrophy. In some cases the first and second sounds are alike and equidistant, showing a short, ill-sustained systole. This is a serious sign, and it is spoken of as "embryocardia." In many cases the first sound is replaced by the murmur of relative incompetence of the mitral valve. Diagnosis is often impossible between idiopathic dilatation with relative mitral incompetence and mitral regurgitation with consequent dilatation. The murmur of mitral stenosis is apt to disappear if dilatation be established. The pulse of dilatation is weak, irregular, and of low tension. It goes to pieces after any physical exertion.

If the right ventricle be in good condition, it will hypertrophy, giving the physical signs of this condition. Especially important is the presence of an accentuated second pulmonary sound and the absence of general venous congestion.

2. *Of Dilatation of the Right Heart.*—If the right ventricle dilate, it will become more enlarged to the right and downward, giving rise to a wavy impulse under the ensiform cartilage and frequently in the six and seventh spaces to the left of the sternum. The accentuated second pulmonary sound is replaced by a weak or absent second sound, and symptoms of venous congestions make their appearance. If the dilatation be extreme, there may be relative incompetence of the tricuspid valve with pulsations of the liver and the jugular veins.

ACUTE ENDOCARDITIS.

Theoretically any part of the endocardium may be the seat of inflammation, but practically the endothelium of the valves and of their immediate vicinity is affected. In postnatal endocarditis the right heart is but seldom involved except in malignant cases. Figures 7–10 show endarteritis and the successive stages of compensation (hypertrophy) and of failing compensation (dilatation).

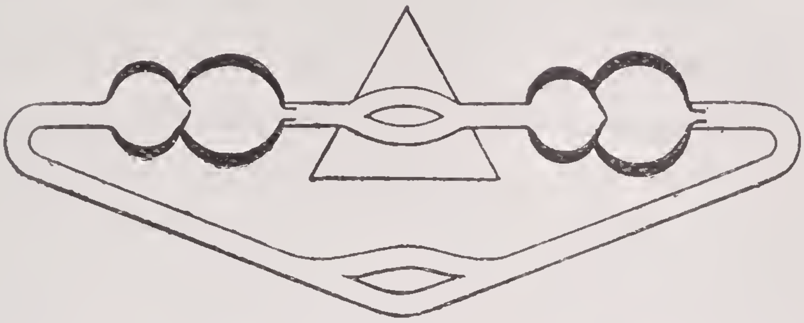


FIG. 7.—Diagram showing normal condition of circulation.

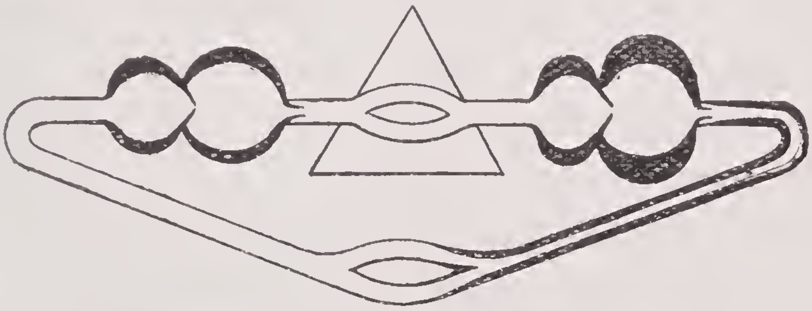


FIG. 8.—Diagram showing compensatory hypertrophy of the left ventricle following endarteritis; right heart normal.

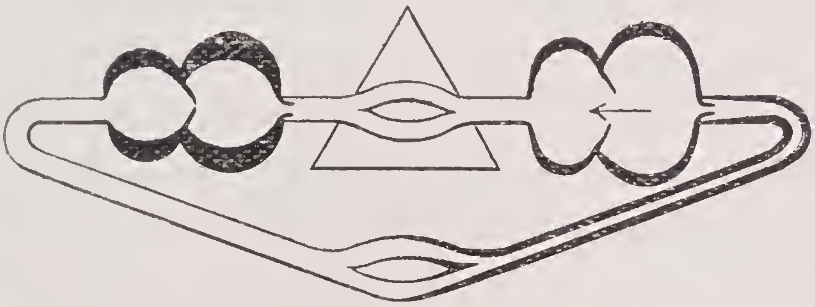


FIG. 9.—Diagram showing failure and dilatation of the left ventricle (failing compensation), stretching of the mitral ring allowing mitral regurgitation, with hypertrophy of the right ventricle (compensatory); some pulmonary congestion, no general venous congestion.

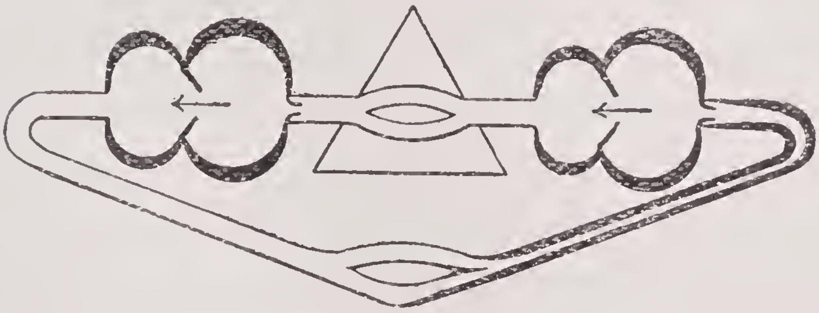


FIG. 10.—Diagram showing total failure of compensation: the right ventricle is dilated; tricuspid leakage occurs; there are both pulmonary and general venous congestions.

Etiology.—Endocarditis does not occur as a primary disease, but always occurs in association with other affections. The affection may be produced artificially by the injection into the blood of various pathogenic micro-organisms, especially if the valves be injured or be the seat of chronic inflammation. The view that endocarditis is a disease of bacterial infection is becoming more and more prevalent. The most important cause is acute articular rheumatism, from 60 to 85 per cent. of all cases being due to this disease. Endocarditis may complicate the abarticular form of rheumatism in children, in which case the primary cause is often overlooked. According to Osler, endocarditis commonly follows chorea: "There is no disease in which post-mortem acute endocarditis has so frequently been found." The disease complicates the exanthemata, especially scarlet fever. It may accompany any septic or infectous disease. It has thus been found with pneumonia, erythema, gonorrhœa, dysentery, pyæmia, and puerperal fever. Suppurative infectious diseases, however, are more commonly the cause of the malignant form. In ulcerative processes such as phthisis, typhoid fever, or ulcerating carcinomata, slight inflammations of the endothelium are frequently found; these have, however, as a rule, no clinical interest.

Pathology.—The endocardium is a connective-tissue membrane covered with epithelium and poorly supplied with blood-vessels, hence its inflammations are of the cellular rather than of the exudative variety. There are three grades of severity: (1) There is a simple swelling of the valve-segments, their surface being smooth. (2) There is swelling of the valves with a new cell-growth in places, so that the surface is covered by vegetations composed of granulation-tissue capped with fibrin. There may be found in the vegetations bacteria of various kinds, but their relation to the disease process is not known. The vegetations occur on the valve-segments at their lines of maximum contact when closed. (3) The cell-growth may be so excessive that the cells undergo necrosis, causing ulceration or perforation of the valve. This condition is more common in the malignant form. The mitral valve is the one most frequently affected, next the mitral and the aortic, more rarely the aortic valve alone.

Effects of the Lesion.—1. The diseased valve is rendered incompetent from stenosis, from insufficiency, or from both.

2. The inflammation may extend to the myocardium, so that it is infiltrated with cells. This inflammation is rarely severe, but it tends to weaken materially the power of the heart.

3. Pieces of vegetations may become detached, enter the blood-current, and lodge in peripheral arteries, causing embolism. The emboli are non-infective in simple endocarditis.

4. In many cases—estimated as one-third by some authors—pericarditis occurs, both diseases arising from a common etiology. Extension of inflammation to the pericardium through the myocardium has occurred, but is exceedingly rare.

5. Acute endocarditis frequently attacks a heart that is the seat of a chronic endocarditis, this condition predisposing toward a new infection, and may thus upset compensation.

6. In acute cases it does not make very much difference which valve is affected. The chief question is, How does it affect the work of the heart? As the lesion is sudden, compensation at first is only by increase of frequency and possibly by increase of force; later comes the question of hypertrophy.

Symptoms.—The symptoms may be divided into three groups: 1. Symptoms of inflammation; 2. Symptoms of valvular insufficiency; 3. Symptoms of embolism.

1. *Symptoms of Inflammation.*—There may rarely be an initial chill. The temperature in almost all cases becomes elevated; it may be 104° or 105° in children, but is rarely as high in adults; it is often irregular.

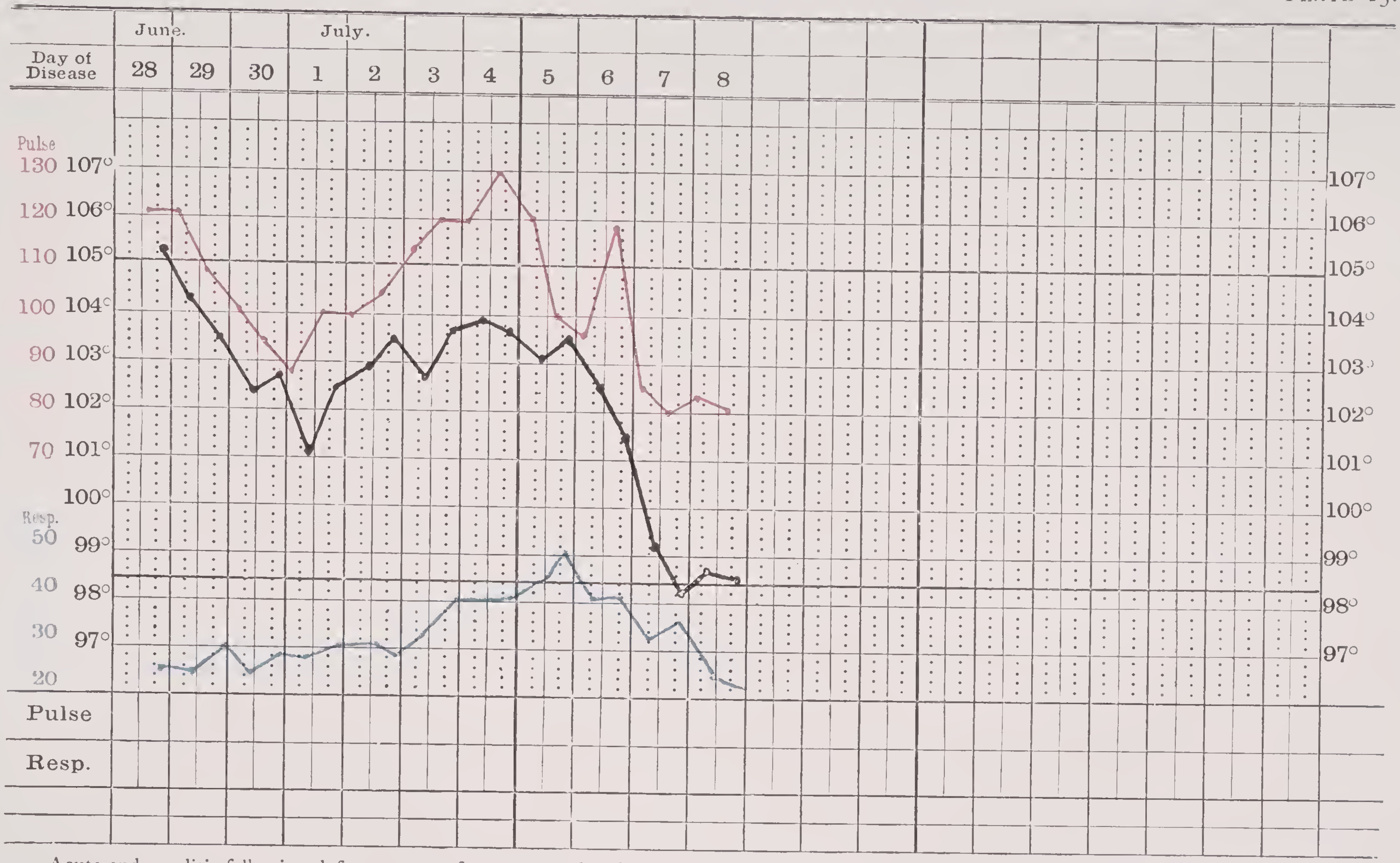
In the majority of cases (75 per cent.) there are peculiar feelings referred to the heart. The patient complains of vague precordial distress, a sense of heaviness, a feeling as if the heart were being squeezed, etc. Actual pain is rare. When it does occur, it is apt to resemble angina and may be referred to the epigastrium. The inflammatory symptoms may be obscured by those of the disease to which the endocarditis is secondary.

2. *Symptoms of Valvular Insufficiency.*—The breathing is rapid, and usually there is subjective dyspnœa. If the myocardium be not involved, the pulse is rapid and the heart's action is tumbling and tumultuous from compensatory over-action. There is the subjective feeling of palpitation. If there be myocarditis, the pulse becomes rapid and feeble. There are symptoms of arterial anæmia—faintness, syncope, small feeble pulse, and spots before the eyes. There may be symptoms of any of the venous congestions mentioned under "dilatation," depending on the severity and extent of the lesion.

3. *Symptoms of embolism* may arise at any time. Sudden lumbar pain accompanied with albuminuria or hæmaturia indicates embolism of the kidney. Pain and swelling of the spleen indicate embolism of that organ. Embolism of the brain is recognized by various paralyses depending upon the location of the embolus, and by disturbances of consciousness. Embolism of the lungs occurs only with endocarditis of the right heart.

Physical Signs.—The most common physical sign is a "bellows murmur," systolic, heard best at the apex and transmitted to the left, although imperfectly. This sign is

ACUTE ENDOCARDITIS.



Acute endocarditis following defervescence of acute articular rheumatism, showing temperature (black), pulse (red), and respirations (blue).

generated by mitral insufficiency. Other murmurs may be heard according to the valve affected, but diastolic murmurs are rare. The rhythm of the murmur is often hard to determine, from the tumultuous action of the heart. The presence of a murmur alone is not diagnostic, and there are cases without murmurs. The association of an over-acting tumultuous heart and a small feeble pulse is of some diagnostic valve. There may be the physical signs of dilatation of the left ventricle with accentuation of the second pulmonary sound. There may be added the physical signs of venous congestion.

Course of the Disease.—1. *Latent Cases.*—The lesion is slight and is perfectly compensated. There are no symptoms. Physical signs may be present. In some of these cases the valve may return to a normal condition. Embolism may occur, however mild the case.

2. *Mild Cases.*—There are present inflammatory symptoms, but the circulation is not disturbed and there are no symptoms of valvular inefficiency. In severer cases there may be a tumultuous action of the heart and some dyspnœa.

3. *Severe cases* result either from an extensive lesion, from a secondary myocarditis preventing compensation, or from an acute endocarditis engrafted upon a chronic process, upsetting compensation. Very rarely there may be sudden death from excessive circulatory derangement. There are developed symptoms of pulmonary congestion. If the right ventricle hold good, there will be no general venous congestions. These, however, will occur if the right ventricle fail in its work.

Prognosis.—Acute endocarditis rarely proves fatal unless complicated by severe pericarditis or myocarditis. A few cases with healthy valves recover, especially those cases secondary to chorea. The liability to organic valvular changes is great. The possibility of repeated attacks, especially in rheumatic cases, must be taken into consideration. An acute attack engrafted upon a chronic endocarditis may upset compensation and lead to a fatal dilatation. Sudden death is exceedingly rare. The occurrence of embolism adds an uncertain element to every case.

Treatment.—The old idea that endocarditis could be prevented by curing rheumatism early has not been sustained. Still, it is best to treat every case of rheumatism and chorea energetically from the very start. Patients with rheumatism should have every source of heart strain removed by as nearly absolute a rest as possible. This does not lessen the patient's liability to have endocarditis, but if it does occur it is much more apt to be less severe, less extensive, and less permanent in its results.

When endocarditis is once established, care should be taken not to depress the heart by too large doses of the salicylates.

Inflammatory symptoms are best treated by strict bodily and mental rest, applications of cold to the heart, and small doses of opium.

The action of the heart should be controlled. Stimulants should be given if indicated. Over-action of the heart may be treated by small doses of aconite or of iodide of potassium, or by the use of cold to the precordia. The bowels should be opened freely, and distention of the stomach is to be avoided.

Venous congestions should be treated by heart stimulants and by depletion by diuretics, diaphoretics, and cathartics. Blood-letting may be employed in selected cases.

The treatment of convalescence must be tonic and supporting, in order that compensation may be made permanent.

MALIGNANT ENDOCARDITIS.

Synonyms.—Septic, Ulcerative, Diphtheritic, Bacterial, Mycotic endocarditis; Arterial pyæmia.

Etiology.—The disease is secondary to a number of septic conditions: (1) It may follow puerperal fever or any septic condition of the puerperal state. (2) It follows septic wounds. (3) It may complicate certain septic diseases. Of these diseases, pneumonia is perhaps the most common. Cases have followed suppurative phlebitis from ear disease, erysipelas, diphtheria, suppurative pylephlebitis, osteomyelitis, dysentery, abscesses, and gonorrhœa. (4) In some cases no apparent cause can be found, but it is supposed that germ infection occurs through unnoticed cracks or abrasions of the skin or the mucous membranes.

The exciting cause of malignant endocarditis is infection of the endocardium by bacteria. There are a variety of micro-organisms capable of causing this infection, the commonest being the cocci of suppuration, the coccus of erysipelas, and the pneumococcus. Infection of the endocardium by bacteria is favored by its previous weakness or inflammation, three-quarters of all cases of endocarditis occurring in hearts previously affected with chronic valvular disease.

Lesion.—The lesion consists in abundant cell-growth of the endocardium, forming vegetations capped with fibrin, the base consisting of granulation-tissue; these vegetations contain colonies of bacteria. The cells are apt to become necrotic, so that by their death there are formed ulcers which may perforate or erode the valve-segments, the septum, or even the heart itself, or which may so weaken the resisting power of the valve that it may become bulged, forming a little aneurysm. There may be infection of the deeper endocardial layers, with the production of small abscesses.

Portions of vegetations containing bacteria are apt to become detached, enter the blood-current, and become emboli of a distinctly infective character. In this way distinct secondary lesions are developed. (1) There may be purulent inflammation of any of the serous membranes, meningitis, pericarditis, empyema, peritonitis, or suppurative inflammation of the joints. (2) There may be suppurative infarctions and abscesses of any part of the body, especially of the brain, lungs, kidney, spleen, and liver. (3) There may be subcutaneous hemorrhages from destruction of the wall of a cutaneous blood-vessel by an infective embolus. Ptomaïnes arising from the growth of the bacteria poison the general system and produce the symptoms of septicæmia.

The location of the inflammation is more widespread in malignant than in simple endocarditis, the right heart and the endocardium lining the heart-cavities being more frequently affected. In 209 cases the aortic and mitral valves together were affected in 41; the aortic valves alone, in 53; the mitral valve alone, in 77; the tricuspid valves, in 19; the pulmonary valves, in 15; the heart-wall, in 33; the right heart alone, in 9. When the endocardium lining the heart-cavities is affected, the most frequent situations are the upper part of the septum of the left ventricle and the postero-external wall of the left auricle. The spleen is large and soft. The cells of the kidneys and the liver show degenerative changes.

Symptoms.—The symptoms may conveniently be divided into three principal groups:

1. *Symptoms of a Sudden and Severe Heart-lesion.*—The symptoms of this group resemble those of simple endocarditis, but are much more sudden and severe. There are marked disturbances of circulation, as shown by venous congestion in both the pulmonary and the systemic circulation. The lesion, as a rule, is too sudden and severe to

allow of any approach to compensation. There are usually murmurs heard according to the valve affected. Diastolic and right-heart murmurs are more common than in simple endocarditis. The action of the heart is often so irregular and tumultuous that the rhythm of the murmurs cannot be determined. In some cases, especially when the lesion is located in the ventricular endocardium, there may be no murmur at all. In three-fourths of the cases there are present the physical signs of antecedent valvular disease. The size of the heart is not increased in acute cases, as the patient is apt to die of sepsis before dilatation can become appreciable; but in subacute cases dilatation may become evident and there may be an approach to a compensatory hypertrophy.

2. *Symptoms of Sepsis.*—There is a decided tendency for the patient to pass into the "typhoid state." There is a fever which is either steady or interrupted by marked remissions. There are frequent chills followed by sudden elevation of temperature, its decline being accompanied by sweating and prostration. Repeated erratic chills indicate septic material in circulation, and they should always cause a diligent search to be made for a septic focus in some part of the body. It should be remembered, however, that patients walking about in a condition of fever are apt to complain of chilly feelings. The chills of suppuration are too erratic to be confounded with those of malarial infection.

3. *Symptoms of Infective Emboli.*—Embolic symptoms give different clinical features according to their localization. Emboli of the brain will produce paralyses of various muscles according to the situation of the embolus, with disturbance of consciousness. There may be abscess of the brain or meningitis, usually associated with furious delirium. In the lungs there may be a septic pneumonia or an abscess. There may be empyema. The spleen may become large

and tender and may be the seat of abscesses. Abscesses in the liver are accompanied by their usual symptoms. Embolism of the kidneys is marked by lumbar pain, by hematuria, and possibly by the presence of pus in the urine. Petechial rashes may resemble the eruption of certain cases of typhoid or of cerebro-spinal meningitis. In some cases, if associated with multiple skin abscesses, the appearance of the patient may be suggestive of hemorrhagic small-pox.

The **diagnosis** of malignant endocarditis is made by the combination of the three groups of symptoms. Any one group, however, may be slight or even latent, and any one group may predominate and give its stamp to the disease.

(1) If the symptoms of the heart-lesion predominate, the case will resemble simple endocarditis or chronic endocarditis with some intercurrent fever, as typhoid, malarial, etc.

(2) If the septic symptoms predominate, the case may resemble one of typhoid fever, surgical septicæmia, or pyæmia. The disease may be mistaken for malarial fever, but the chills are too erratic, the temperature does not yield to quinine, and the blood-examination does not reveal the malarial organism. The disease may also resemble acute miliary tuberculosis, but a detailed examination of the case and the finding of the bacilli in the sputa will render the diagnosis easy.

(3) If the embolic symptoms predominate, the case may resemble non-infective embolism from simple endocarditis, acute or chronic, or the secondary suppurative inflammations may resemble those of primary origin. Thus the case may be mistaken for meningitis, for abscess of the brain, for empyema, etc.

Duration.—Some cases run an acute course of about two weeks' duration. Death results from the derangement of the circulation due to the heart-lesion, from sepsis, or from the infective emboli. Other cases run a subacute course of from ten to twelve weeks, and more rarely may continue for six or eight months. In these chronic cases the infection is less severe, the damage to the heart is less extensive, and there is usually an attempt at compensation. A few of these chronic cases recover, especially if compensation be perfected and if embolism does not occur.

The **prognosis** is generally bad. Only a few cases recover. These cases are those which occur most frequently after puerperal infection and which have run a chronic course without embolism. These cases, however, are left with permanently disabled valves.

Treatment.—The treatment is that of both the heart inflammation and the septic condition. For the heart condition the treatment is that of ordinary endocarditis, but carried out more vigorously, and heart stimulants are more commonly indicated.

There are two ways by which the septic condition may be treated. One method is by the administration of quinine in large doses, so that the patient is kept thoroughly cinchonized. The unpleasant effects of quinine being mitigated by adequate doses of phenacetine or sodium bromide, from 20 to 40 grains of the drug may be given daily. The second method consists in the giving of alcohol in large doses, so that the patient is kept continuously under its influence.

The secondary suppurations are to be treated on general surgical principles.

CHRONIC ENDOCARDITIS; VALVULAR DISEASE.

The terms "chronic endocarditis" and "valvular disease" are practically synonymous, as it is the endocardium of the valves that is almost regularly affected. It is well to distinguish between two sets of cases, (1) chronic endocarditis proper and (2) atheroma.

CHRONIC ENDOCARDITIS.

Pathology.—The lesion consists in the thickening of the valve by increased cell-growth and the formation of firm connective tissue. In this way the valve-segments become contracted, deformed, and insufficient, producing either stenosis or insufficiency, or both. Lime-salts may be deposited in the thickened valve, so that it may become a dense calcareous mass with hardly a vestige of normal tissue. There may appear vegetations and ridges formed by irregular growth of cells and connective tissue, and upon these ridges fibrin may be deposited. Detachment of the fibrin in masses will give rise to emboli which are simple and non-infective in this disease. The proliferated cells in the valve may be the seat of fatty degeneration, producing opaque yellow spots frequently infiltrated with salts of lime, or the fatty cells may break down, so that little superficial ulcers result. To the combination of chronic connective-tissue proliferation and fatty degeneration of the cells the name "atheroma" is applied.

Etiology.—Chronic endocarditis regularly follows an attack of acute endocarditis produced by any of its causes. In some cases, however, it is difficult to obtain a clear clinical history of the primary attack. About half the cases have a rheumatic origin. The disease is most frequent in children and young adults, and affects the mitral valve with the greatest frequency.

Symptoms.—(1) There are symptoms due directly to the diseased valves—aortic stenosis or regurgitation, or mitral stenosis or regurgitation. Involvement of the valves of the right heart is exceedingly rare. (2) There is developed compensatory hypertrophy; or (3) a dilatation with various venous congestions due to the enfeebled pumping power of the heart. (4) There may be symptoms of embolism.

ATHEROMA.

In this class of cases the endocarditis is chronic from the start.

Pathology.—The lesion in the valves is the same as that of chronic endocarditis, but is only part of a general set of lesions. There is atheroma of the aorta, which may be dilated. The small arteries show the lesions of chronic endarteritis; their walls are thickened by connective tissue and may be infiltrated with lime-salts; their lumen is narrowed. In the small arteries of the brain miliary aneurysms may be formed. There may be spasm of the peripheral arteries. The lungs are frequently emphysematous. The kidneys usually show the lesions of the atrophic form of chronic diffuse nephritis. There is frequently cirrhosis of the liver. These changes are spoken of under the general name "arterio-capillary fibrosis," and will be described with more detail in a later heading. The aortic valves are the favorite seat of the lesion. The mitral valve may also be involved, but it is rarely involved alone.

Etiology.—Atheroma is a disease of adult life, few cases being seen before the fiftieth year. It is more common in men than in women, and is especially frequent in those who lead a life of exposure, intemperance, and severe muscular strain. It is thus common in longshoremen. Alcoholism, syphilis, gout, chronic rheumatism, and chronic lead-poisoning are cited as causes, and there seems to be a distinct family predisposition toward this series of degenerative changes.

The **symptoms** are in the main those of the first class, but they differ in the following respects:

1. The lesion is not only of the heart, but of other organs as well, more work being thrown on the diseased heart by reason of the increased peripheral resistance in the

aorta and the peripheral arteries, and the arterial spasm due to the kidney disease.

2. The lesions occur in late adult life, when recuperative powers are on the wane and when compensatory hypertrophy is imperfect at the best.

3. The patients are usually persons leading an intemperate life with severe muscular strain, and unwilling—indeed, unable—to take the proper care of themselves; by reason of their condition of life they are poor subjects for compensatory processes.

4. The lesion is progressive. The valves are not only deformed, as in the first class, but tend to become more and more involved.

Detailed symptoms of both sets of cases will be given under the head of the individual valvular lesions.

Mitral Incompetency.

Etiology.—Insufficiency of the mitral valve results from one of three causes: (1) Contraction or shortening of the valve-segments from chronic endocarditis, frequently associated with changes in the chordæ tendineæ and with more or less narrowing of the orifice. (2) Dilatation of the mitral ring from dilatation of the left ventricle. (3) Defective muscular closure from myocarditis, or from the weakening of the heart muscle in anæmia and prolonged fever.

It is important to remember that mitral insufficiency is not always a sign of chronic endocarditis, but that the valve-segments may be normal, the incompetency being "relative."

Pathology.—As the result of the regurgitation past the mitral valve, at each systole of the ventricle the left auricle receives blood from two sources—its regular supply from the pulmonary veins, and the abnormal supply from the left ventricle. The auricle therefore becomes dilated and to some extent hypertrophied, although the latter process is never well marked in the case of an auricle. At the time of the diastole of the left ventricle this abnormal amount of blood at high pressure pours into it, over-filling it. To accommodate this increased amount of blood the ventricle must necessarily dilate. Although at the time of the ventricular systole only part of the blood is pumped forward in the direction of the normal blood-current, the remainder being forced back into the auricle, still, to get rid of the large supply of blood, the work of the left ventricle becomes excessive, and therefore the ventricle becomes hypertrophied. Owing to the over-filling of the auricle during diastole, the pulmonary veins are less readily emptied; the right ventricle expels its contents less readily, and becomes both dilated and hypertrophied. If the hypertrophy of the right ventricle is adequate to maintain the equilibrium of the pulmonary circulation, there are no signs of venous congestions in the systemic circulation. If compensatory hypertrophy fails, however, then general venous congestions will ensue.

Congestion of the pulmonary vessels is not, as a rule, as marked in mitral regurgitation as in mitral stenosis, because the back pressure is more intermittent, occurring only during diastole, whereas in mitral stenosis it is continuous.

Cases of relative incompetence due to impaired nutrition of the cardiac wall or to its dilatation are not usually well compensated. There is apt to be considerable engorgement of the pulmonary vessels, with bronchitis and often with fairly profuse hæmoptysis, and, from general failure in the power of the right ventricle to work in face of the pulmonary back pressure, general venous congestions will ensue.

Symptoms.—If compensation is good, there may be no symptoms noticed by the patient even for years. There may, however, be some palpitation and dyspnœa on exertion, with a bluish tinge to the lips and the ears. Attacks of bronchitis or of hæmoptysis may occur.

If *compensation fails*, the symptoms of pulmonary engorgement become more marked; there are palpitation, weak and irregular action of the heart, steady dyspnœa with developing orthopnœa, increase of cough with bloody or watery expectoration, and dropsy, first in the feet and then becoming more generally distributed.

By judicious treatment compensation may again be established, and the patient will recover from the attack. Subsequent attacks grow more frequent and severe, and recovery from them becomes less and less satisfactory, until at last a permanent condition of general dropsy and venous congestions results, terminating the life of the patient. Sudden death is exceedingly rare.

Physical Signs.—The characteristic murmur of mitral regurgitation is systolic in rhythm, is heard with maximum intensity at the apex, and is convected into the left axilla and the back. The murmur, which may be loud enough to be heard over the whole of the chest, is usually of a blowing, puffing character, but it may have a musical quality. The character of the murmur gives no indication of the degree of the insufficiency. The murmur may come and go, and when absent it may frequently be reproduced by the upright position, by deep respirations, or by exertion. There are cases in which the murmur has its maximum intensity along the left border of the sternum, at the level of the second or third rib, where the dilated auricle approaches

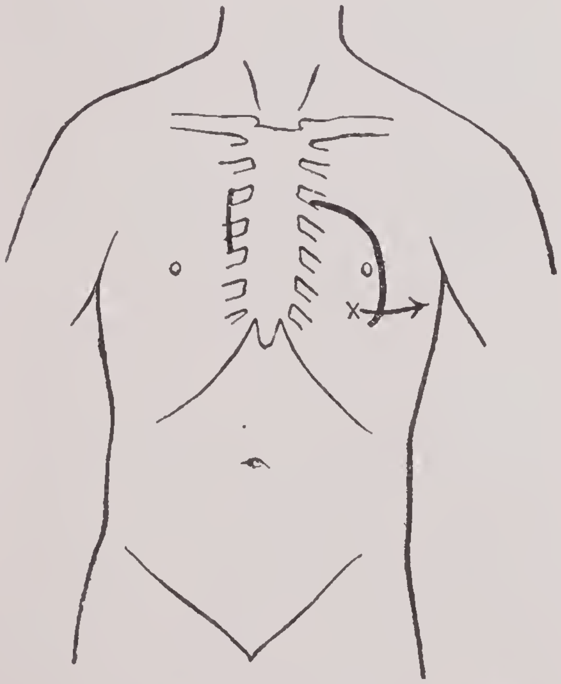


FIG. 11.—Mitral regurgitation, showing the area of cardiac dulness, the point of maximum intensity of the systolic murmur, and the direction in which it is carried.

the chest-wall. There may also be heard the rumbling or purring presystolic murmur of an associated mitral stenosis. In some cases the presystolic murmur alone is heard, even if there be regurgitation as well. A systolic thrill is often appreciable at the apex, but this sign is not of much diagnostic value.

The second pulmonary sound is accentuated in almost all the cases where there is compensatory hypertrophy of the right ventricle. This sound is best heard in the second interspace to the left of the sternum.

There are present the ordinary physical signs of hypertrophy of the left ventricle, and usually of the right ventricle as well, or, in case of failing compensation, the signs of their dilatation.

The pulse may show nothing abnormal, or it may be irregular. There is no characteristic sphygmographic tracing.

The **diagnosis** should be made, not from the presence of the murmur alone, but from the other signs as well—the accentuated pulmonary second sound, the enlargement of the heart, and the clinical history. Mitral incompetency should be distinguished in this manner from those hæmic murmurs which are heard at the apex of the heart, and from the so-called "lung heart" or the Potain-Rosenbach murmur. This murmur is generated in the overlying lingula of lung by the pressure of the heart's impulse against a small bronchus. By this compression at each systole during inspiration a systolic puffing sound is produced. This sound, however, is not transmitted to the left, and is heard only during inspiration.

The diagnosis of relative incompetence due to anæmia or to exhausting disease should be made by a careful review of the case. The diagnosis is necessary not only in point of prognosis, but also in governing the treatment.

Prognosis.—Mitral incompetency is perhaps the least serious of all the valvular affections, as it usually occurs in young subjects, in whom compensatory hypertrophy is possible. The prognosis of relative incompetence due to anæmia or to exhausting disease is good if the primary disease can be cured. The cases which develop in consequence of dilatation of the left ventricle usually do badly, as the dilatation generally precludes all idea of compensation.

MITRAL STENOSIS.

Etiology.—Mitral stenosis, except for rare congenital cases, is always the result of valvular change. The affection regularly follows a previous attack of endocarditis, is usually seen in early life, and is more common in women than in men, in the proportion of 4 to 1, because girls are more liable than boys to rheumatism and chorea. The onset of the disease is often so insidious that its origin cannot always be determined.

Pathology.—The valve-segments may be stiffened into a rigid mass, or they may be fused together, forming a conical opening, the "funnel-shaped mitral." The orifice of the valve may be constricted to form a narrow slit, the "buttonhole mitral," or it may be so constricted as to admit only the very tip of the little finger.

A stenotic mitral valve is almost always incompetent at the same time. The effect of the stenosis is to impede free passage of blood from the left auricle into the ventricle, causing thus steady back pressure, from which other changes in the heart result.

The left auricle becomes much dilated and hypertrophied, its muscular walls being increased from two to four times in thickness. The over-filled auricle impedes the outflow of blood from the pulmonary veins; pulmonary engorgement results, being more marked in this than in any other valvular affection. The majority of cases of pigment induration of the lungs are due to this condition of engorgement. Dilatation and compensatory hypertrophy of the right ventricle result, compensating for the increased tension in the pulmonary vessels and equalizing the lesser circulation. In course of time, when the right ventricle fails in maintaining its power and hypertrophy, it will weaken and dilate, relative incompetence of the tricuspid valve will ensue, and general venous congestions will presage a fatal issue.

In uncomplicated mitral stenosis less than the normal amount of blood enters the ventricle to be pumped into the arteries; hence less work is required of the ventricle, and neither dilatation nor hypertrophy should occur. In cases, however, associated with incompetency of the valve, hypertrophy or dilatation of the ventricle occurs. In rare cases the ventricle may hypertrophy without any appreciable cause. It is supposed that increased peripheral resistance from general contraction of the arteries, caused by their irritation by imperfectly-oxidized blood owing to pulmonary congestion and engorgement, might account for these cases.

The **symptoms** of mitral stenosis resemble those of mitral regurgitation, both conditions producing the same results—arterial anæmia and venous congestions, first in the pulmonary system, later in the systemic veins when failure of the right ventricle occurs. Symptoms of pulmonary congestion are, however, more marked and constant in stenosis than in insufficiency. Children with mitral stenosis are usually poorly developed.

Physical Signs.—The cardiac impulse is often most appreciable in the region of the lower sternum and in the fourth and fifth left interspaces, being caused by the hypertrophied and dilated right ventricle approaching the chest-wall in these situations. Localized just above and within the apex may be felt a distinct vibratory or "cat's-purr" thrill. This thrill is presystolic and terminates with a sudden sharp shock synchronous with the cardiac impulse. When present, it is pathognomonic of mitral stenosis. Care should be taken not to mistake this apex thrill for a diastolic thrill at the base due to aortic regurgitation.

The murmur of mitral stenosis is heard to the inner side of the apex beat over a limited area (Fig. 12), and is not transmitted in any direction. Its rhythm is presystolic, or, more properly speaking, *auricular-systolic*, as the murmur is presystolic only as regards the ventricular systole. The murmur is a rough, rolling, purring sound, represented by pronouncing "rup" or "r-r-r-rup," having in the latter case a rolling drum-beat character. It may terminate abruptly with the first sound, which is unusually clear and snappy, or there may be a distinct interval between the sounds. The murmur may consume a good part of the diastole, or it may be heard only during the latter part of it. The murmur often comes and goes, usually disappearing if compensation fail, only to reappear should compensation again be established. It is often heard better with the ear than with the stethoscope.

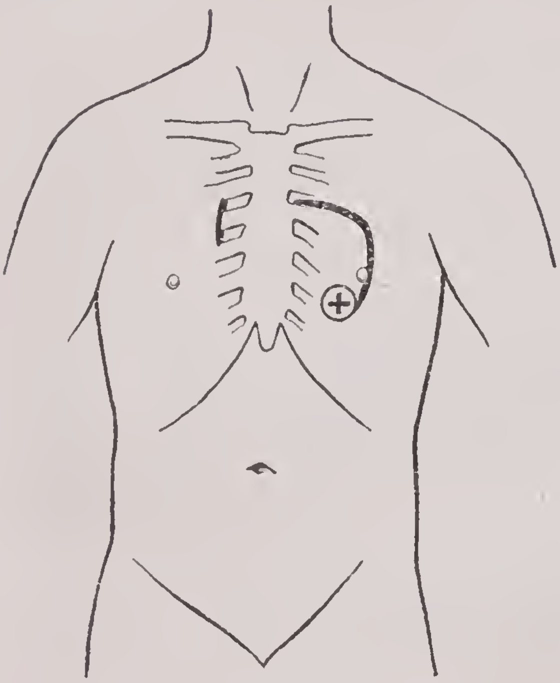


Fig. 12.—Mitral stenosis, showing area of cardiac dulness, location of the presystolic murmur, and the area over which it is heard.

If regurgitation coexist with stenosis, there will also be heard the murmur of the former affection ; this murmur may be so faint as to be heard only when the breath is held.

Valuable evidence is afforded by the second pulmonary sound. This sound, which is sharply accentuated if the right ventricle is doing its work well, is reduplicated in about one-third of all cases, and its reduplication is strong presumptive proof of mitral stenosis. The second aortic sound is weak, as the amount of blood entering the aorta is insufficient to raise its tension.

Hypertrophy and dilatation of the left auricle and the right heart, and possibly of the left ventricle as well, give their customary physical signs.

The pulse of mitral stenosis is small as compared with the action of the heart, the arteries being under-filled.

A characteristic of mitral stenosis is the occurrence of interpolated beats in the line of the descent of the pulse-wave. These interpolated beats, which are well seen in the accompanying sphygmographic tracings (Figs. 13, 14), are little abortive systoles, started as extra contractions of the overloaded auricles and communicated thence to the ventricles. As their irregular systoles occur during the time of

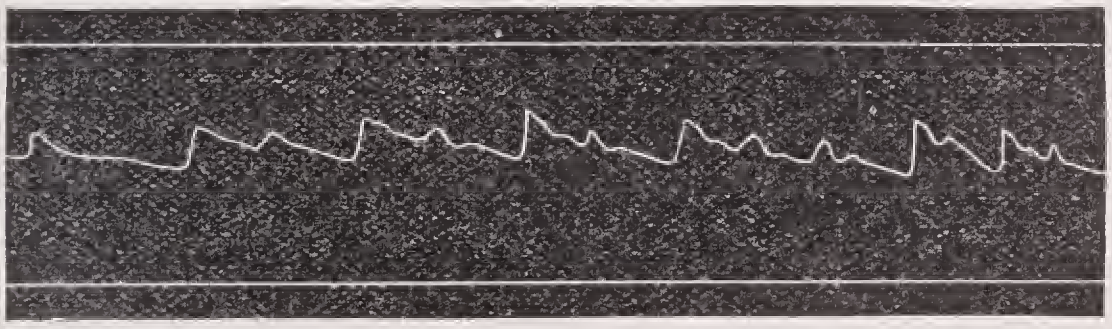


FIG. 13.—Sphygmogram showing the interpolated beats of mitral stenosis.

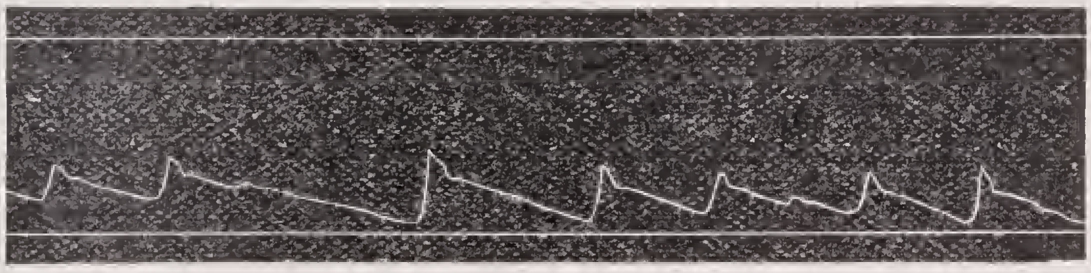


FIG. 14.—Sphygmogram showing the interpolated beats of mitral stenosis.

diastole, when the ventricle is under-filled with blood, a distinct and well-marked pulse-wave cannot be formed.

Diagnosis.—The murmur of mitral stenosis may be mistaken for the diastolic murmur of aortic regurgitation, transmitted down to the apex and heard there late in the diastole. Examination at the base of the heart should reveal the maximum intensity of the aortic murmur. The character of the pulse should assist in the differential diagnosis. The diagnosis is more difficult if the murmur be absent. The diagnosis must be made upon the accentuated or reduplicated second pulmonary sound, the weak second aortic sound, the sudden, snappy first sound at the apex, and the physical signs of enlargement of the right ventricle.

The **prognosis** in mitral stenosis is not so good as that in mitral insufficiency, because the back pressure of the pulmonary veins is constant and not intermittent, and because the force of the left ventricle cannot be called into requisition to aid in the compensation of the lesion.

Aortic Regurgitation.

Etiology.—Insufficiency of the aortic valves may be caused by the following conditions:

1. *Congenital lack of development.*

2. *Rupture of a valve-segment.* A healthy valve-flap may in rare instances be caused by excessive strain, such as heavy lifting; or an ordinary strain may be the means of rupturing a valve that is weakened by ulcerative changes.

3. *Stretching of the aortic ring*, causing "relative incompetence." This condition is rare, and is seen only in cases of extensive atheroma of the aorta with great dilatation just above the valves.

4. *Acute endocarditis.* Aortic incompetence does not result during an acute attack unless the valve be eroded or ulcerated. It is more commonly seen, therefore, in malig-

nant endocarditis. Slow changes, however, may result in the shrinkage, contraction, and calcification of the valve, causing it to become incompetent. Aortic incompetency may thus be seen in children with antecedent history of rheumatism and acute endocarditis, but it is not so common under these circumstances as mitral disease.

5. By far the most common cause of incompetence is the slow contraction due to atheroma, seen in able-bodied laborers who are subject to heavy muscular labors and who over-indulge in alcohol. There may be a syphilitic element which of itself is capable of causing arterial sclerosis. Occurring as the result of atheroma there are apt to be found associated lesions in the aorta, arteries, kidneys, liver, and lungs, already alluded to (see Atheroma, page 225).

Pathology.—As the result of the incompetency of the aortic valve, blood flows from the aorta back into the ventricle during the diastole. The left ventricle then receives blood from two sources—the normal supply from the auricle, and the regurgitated blood from the aorta. The ventricle therefore becomes greatly dilated. Dilatation is all the more extreme because the distention of the ventricle occurs during diastole, at which time the tissues are in a relaxed condition, and also because the heart-wall is often the seat of fatty degeneration, as will be shown hereafter. The increased labor of expelling this large amount of blood, part of which is to roll back again, leads to hypertrophy of the left ventricle. This hypertrophy reaches the highest degree seen in any valvular disease, and may produce a heart of enormous size and weight (from 30 to 50 ounces), to which the name "bovine heart," or *cor bovinum*, has been applied. This is especially the case in children.

Relative incompetence of the mitral valve is common as the result of the dilatation of the left ventricle; when this incompetence occurs there is apt to be pulmonary congestion with compensatory hypertrophy of the right ventricle.

There is a tendency in aortic regurgitation for the heart to undergo fatty or fibroid degeneration from poor coronary circulation, either because of the associated atheroma or calcification of the coronary arteries, diminishing their calibre, or because the coronary arteries, by reason of the diminished tension in the aorta, are poorly filled with blood.

Aortic regurgitation is often associated with aortic stenosis, but regurgitation alone is more common than stenosis alone. Aortic aneurysm may complicate the valvular disease. In advanced cases there may be changes in the cardiac nerves and ganglia that may lead to angina pectoris.

Symptoms.—As long as the hypertrophy equalizes the valvular defect there are no characteristic symptoms. In advanced cases with myocardial degeneration or with lesions in the aorta and coronary vessels there are apt to be developed symptoms of *arterial anæmia*—headache, dizziness, irritability of temper, faintness even to the point of syncope, palpitation, dyspnœa on exertion, with the general symptoms of anæmia. There may be dull aching pain in the precordium, or else attacks of angina pectoris. If at any time the diastole be unduly prolonged, the regurgitating blood may so empty the aorta and large vessels as to cause sudden cerebral anæmia. Sudden death may occur under these circumstances, and its possibility must always be considered in making the prognosis. Œdema of the feet and dyspnœa with progressing symptoms of venous congestion usher in a fatal issue, and differ in no essential features from the venous congestions and heart failure of other valvular lesions.

The **physical signs** of aortic regurgitation are apt to be clean-cut and distinctive. The characteristic murmur, which is diastolic in rhythm, replacing the second sound, is usually heard best in the mid-sternum, at the level of the third rib, and is convected downward toward the lower end of the sternum and the apex. It may be heard best in the second right interspace or at the lower end of the sternum, or even just within the apex. If heard in these latter localities, to which it has been convected downward, it may closely resemble the presystolic murmur of mitral stenosis. Aid to diagnosis in such cases is afforded by the presence of the murmur at the base of the heart as well, and by the other physical signs. Often the murmur is better heard with the ear than with the stethoscope. It may be harsh and of a "sawing" character, or it may be a soft, long-drawn bruit. It is very constant and reliable.

The first sound at the apex is usually weak, and may be replaced by the murmur of relative incompetence of the mitral valve.

The first sound at the base may be replaced by a murmur. This may mean stenosis of the aortic valve, or merely roughening of the surface of the valve or of the intima of the aorta just above the valve.

There may be a distinct diastolic thrill over the base of the heart. This thrill may be so diffused as to reach the apex and be then mistaken for the thrill of mitral stenosis; but it is not limited to the apex, nor does it terminate with the sharp shock of the cardiac impulse, as does the thrill of mitral stenosis.

The character of the pulse gives material aid in diagnosis. There is visible pulsation of the peripheral arteries, even in the vessels in which pulsation is not normally visible. The arteries may appear tortuous, straightening themselves with a peculiar jerky motion with each systole. There may be capillary pulsation under the finger-nails or over any skin area artificially reddened by friction. However common this sign may be in aortic regurgitation, it is also seen in profound anæmia, in neurasthenia, and in conditions associated with great relaxation of the peripheral arteries, and hence is not in any sense pathognomonic.

There may be pulsation in the second right intercostal space or in the suprasternal notch that may lead to the diagnosis of aneurysm. There may be a diastolic pulsation of the liver, even if the tricuspid valve be competent. Ophthalmoscopic examination reveals visible pulsation of the retinal arteries of a characteristic jerking quality.

There is heard a to-and-fro murmur in the femoral artery. On palpation the characteristic " water-hammer " or " Corrigan " pulse is felt. The pulse strikes the finger with a sudden forcible impulse, and then at once collapses, leaving the artery empty. This is best appreciated at the radial artery when the hand is held above the head. The quality of the pulse is plainly recognized in the sphygmographic tracing (Fig. 15).

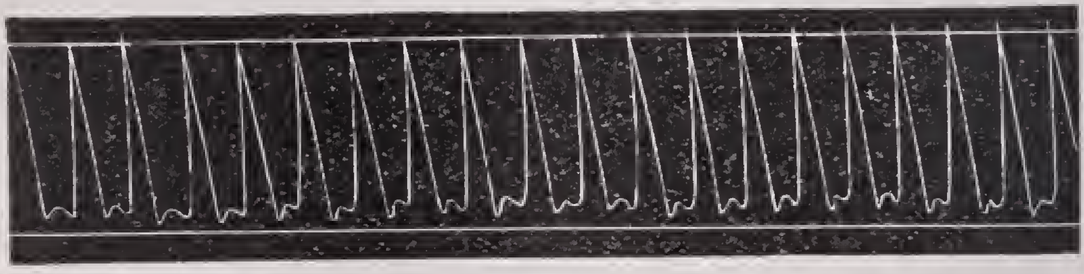


Fig. 15.—Sphygmogram of aortic regurgitation.

Associated with these characteristic signs of aortic regurgitation are those depending upon the increased size and muscular power of the heart. There is a wide forcible area of cardiac impulse, the apex beat often being in the sixth or seventh interspace, and being perhaps as far displaced as the anterior axillary line. There may be bulging of the precordium in children. This increase of size, also determined by percussion, is due to dilatation and hypertrophy of the left heart, and possibly of the right heart as well.

The **prognosis** in aortic regurgitation is not good, for three reasons: (1) As the affection occurs usually in elderly overworked alcoholic subjects as a degenerative change frequently associated with arterial and renal lesions, compensation is neither complete nor sustained. (2) From the frequent complication of fatty degeneration of the heart-muscle, there is a tendency to sudden or gradual heart failure. (3) There may at any time be a sudden over-distention of the ventricle, causing its paralysis, and the sudden death of the patient from acute cerebral anæmia. The liability to sudden death should always be remembered in giving the prognosis.

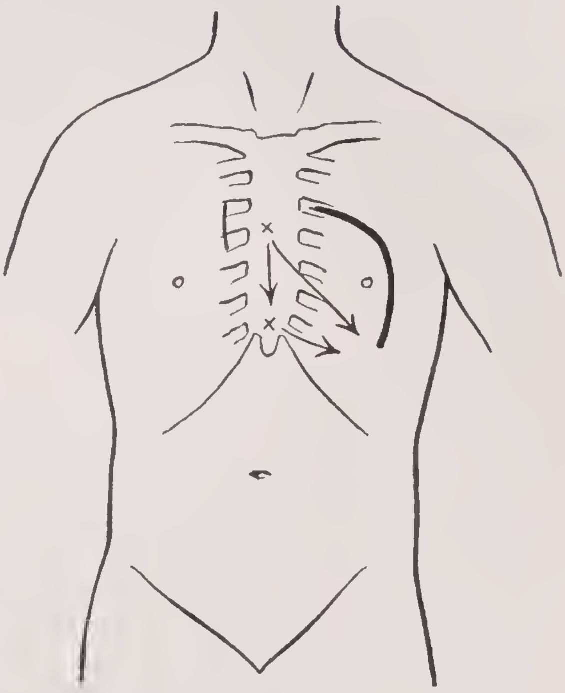


FIG. 16.—Aortic regurgitation, showing the area of cardiac dulness, the usual points of the maximum intensity of the diastolic murmur, and the direction in which it is carried.

AORTIC STENOSIS.

Etiology.—The lesion of aortic stenosis may be the result of chronic endocarditis following an acute attack causing thickening and rigidity of the valve. Usually, however, the disease occurs in old people as an atheromatous change, and is associated with some degree of incompetency. The latter condition is the more serious, and stamps the disease with its own characteristics.

Pathology.—The valve-segments may be simply adherent to each other, or they may be thickened, contracted, or calcified. There may be a tongue of fibrin or large vegetations projecting into the orifice, further obstructing it. To overcome the obstruction to the onward passage of blood through the aortic outlet, more force is required of the left ventricle. It consequently hypertrophies. Usually there is but little dilatation. The whole force of the ventricle being thus called into requisition, compensation is usually good and the remaining parts of the heart are not affected. It is only when the left ventricle begins to fail that there is dilatation of the auricle, impeded pulmonary circulation, and increased work for the right heart.

Symptoms.—There are no symptoms characteristic of aortic stenosis. The affection may last for years and be discovered finally by accidental examination. In advanced cases, where a lessened amount of blood enters the aorta with each systole, there may be symptoms of anæmia, such as dizziness, faintness, and spots before the eyes. In more advanced cases there may be Cheyne-Stokes breathing during the latter part of the disease. When compensation fails the symptoms of pulmonary and systemic congestion do not differ in any way from those caused by other valvular affections.

Physical Signs.—The characteristic murmur is systolic, heard best in the second right interspace, and is conducted upward along the course of the great vessels. Such a murmur is not distinctive of aortic stenosis, as it may be caused as well by simple roughening of the aortic valve or of the intima of the aorta above the valve, or by anæmia. If due to stenosis, the murmur is frequently harsher than if due to the other causes, but even then it may become faint and distant if the left ventricle begin to fail. The second sound at the aortic area is usually weak from diminished blood-pressure in the aorta at the time of the diastolic closure of the valve. This sign may be of great aid in diagnosis. In other cases the second aortic sound is replaced by the murmur of aortic regurgitation. There is frequently at the base a systolic thrill which may be very well marked. There are the ordinary physical signs of hypertrophy of the left ventricle, and, in the later stages, of its dilatation, and with the dilatation the enlargement of the right heart from hypertrophy or dilatation. The pulse is small in size, is regular in rhythm, and may be somewhat slow.

Prognosis.—In simple stenosis the prognosis is generally good, as compensation is easily accomplished by hypertrophy of the left ventricle. If the stenosis be accompanied by regurgitation, the prognosis will depend upon the latter condition.

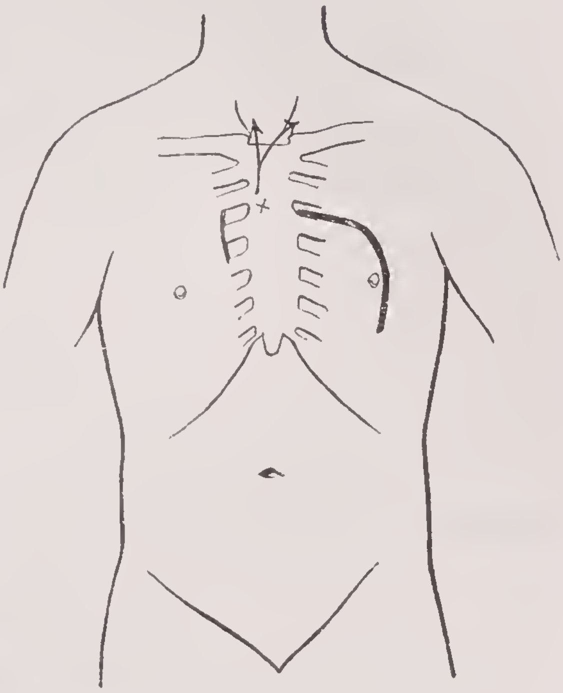


Fig. 17.—Aortic stenosis, showing the area of cardiac dulness, the point of maximum intensity of the systolic murmur (×), and the direction in which it is carried.

Tricuspid Regurgitation.

This condition may result as an acquired affection in endocarditis, especially in the malignant form.

Relative incompetence is far more common, and is due to dilatation of the right ventricle with stretching of the tricuspid ring, or to poor muscular contraction of the ventricle. It is thus produced by a failing left heart, and by any cause producing obstruction in the pulmonary circulation, such as emphysema and interstitial pneumonia. In either case it is a consequence of failure in compensation of the right ventricle. When tricuspid regurgitation occurs, the blood at the time of systole regurgitates from the right ventricle into the auricle and the veins, with the production of venous congestions.

The **physical signs** of tricuspid regurgitation are—(1) A systolic murmur, usually low and soft, heard with maximum intensity at the lower part of the sternum, and transmitted to the right, frequently as far as to the axilla. (2) Enlargement and fulness of the jugular veins. (3) A venous pulsation in the superficial veins of the neck, and frequently a pulsation of the liver. This latter sign is best made out by bimanual palpation, and should not be confounded with the apparent pulsation imparted to the liver by an over-acting right ventricle. (4) Marked increase of general venous congestions.

Tricuspid Stenosis.

Congenital cases of tricuspid stenosis are not uncommon. The acquired form occasionally occurs, usually associated with lesions of the left heart, especially with mitral stenosis.

As the only means of compensation is by hypertrophy of the relatively weak right auricle, effectual compensation cannot occur. Marked venous congestions with great cyanosis are the inevitable result.

The **physical signs** of tricuspid stenosis are—(1) A presystolic murmur heard at the base of the ensiform cartilage. (2) Hypertrophy and dilatation of the right auricle. (3) Occasionally a presystolic thrill over the lower part of the sternum.

Pulmonary Regurgitation.

This affection, which is almost invariably the result of congenital malformation, is exceedingly rare. The regurgitation of blood backward into the right ventricle is followed by great dilatation, and relative incompetence of the tricuspid valve is very apt to result.

Compensation is necessarily imperfect, and a fatal issue is not long delayed.

The **physical signs** of pulmonary regurgitation are—(1) A diastolic murmur heard in the second left interspace, and convected downward and to the right. It is difficult to differentiate the murmur from that of aortic regurgitation. (2) Enormous hypertrophy and dilatation of the right ventricle. (3) The physical signs of the relative incompetence of the tricuspid valve.

PULMONARY STENOSIS.

This affection, which is of great rarity except as the result of disease or of arrested development during intra-uterine life, is one of the commonest forms of congenital malformations, and is often associated with an open foramen ovale or an imperfect interventricular septum. As an acquired disease it may result from malignant endocarditis.

There is considerable hypertrophy of the right ventricle, with dilatation, but compensation is seldom perfect, being easily upset by intercurrent pulmonary affections. The dilatation of the right ventricle allows of tricuspid regurgitation (relative incompetence) in the majority of cases.

The **physical signs** of pulmonary stenosis are—(1) A systolic murmur heard in the second left interspace, and convected a short distance upward and to the left. (2) A weak or absent second pulmonary sound. (3) Marked hypertrophy and dilatation of the right heart. (4) There may be the murmur of relative incompetency of the tricuspid valve, with fulness and possibly pulsation of the superficial veins, especially those of the neck.

Treatment of Chronic Valvular Disease.

The treatment of chronic valvular disease may be divided into that of the stage of compensation and that of its failure.

Stage of Compensation.—Hypertrophy is in itself compensatory of valvular defects, and if the circulation be maintained by it so that the arteries are kept filled and the venous flow is not obstructed, there is no medicinal treatment necessary. Much harm is done by injudiciously prescribing digitalis whenever a murmur is heard, no regard being paid to whether the lesion is compensated or not. Still, it is necessary that compensation should be maintained. The balance between the available power of the heart and the work required of it may be so delicate as to be upset easily by weakness on the one side or by increase of work on the other. Should the patient run down by reason of old age, sickness, or vicious habits, the myocardium will become degenerated and its power will be weakened, while, on the contrary, the hypertrophy may be inadequate to meet any demand for increased work.

1. *The patient should be kept in good health.* To secure intelligent co-operation it may be necessary to inform the patient of the lesion, although it is usually best to confide in some intimate friend or member of the family, upon whose judgment reliance can be placed. The patient should lead a quiet, orderly, and well-regulated life. The diet should be simple and wholesome, and all digestive errors are to be corrected by appropriate measures. Tobacco, tea, and stimulants should be avoided. Turkish baths should be eschewed, and the patient should not live in too high an altitude. Mental worry, over-fatigue, and severe bodily exposure should be avoided. As the prognosis of valvular disease with compensation is much better than was formerly supposed, and as sudden death occurs only with aortic regurgitation, the patient, if informed of his complaint, should be so encouraged and stimulated as to dispel mental depression and despondency.

2. *The work thrown on the heart should be lessened as*

much as possible. In almost all cases enough exercise should be taken to keep the general health good. In fact, the heart's power may even be developed by graduated exercise. Oertel recommends ascending hills of increasing steepness and length until compensation is fully established. At no time, however, should exercise or work ever be allowed to pass to the point of excessive fatigue, nor should sudden violent exercise be permitted. Mental excitement of all kinds should be interdicted.

The condition of arterial tension should always be determined. Should it be raised, pointing to increased peripheral resistance, it should be reduced and kept reduced.

The diet should be simple; over-eating and drinking are to be checked; the bowels must be kept open and diuretics be administered. These procedures may suffice without the need of drugs. Should the latter be indicated, iodide of potassium (gr. x t. i. d.), chloral hydrate (gr. v to vii t. i. d.), and nitroglycerin (gr. $\frac{1}{100}$ q. 3 h.) are of the greatest value.

Treatment of Failing Compensation.—The treatment naturally is to be directed to fulfil four indications:

1. *To lessen the work required of the heart.*

(*a*) *By rest.* This of itself may restore disturbed compensation, and should be resorted to in all serious cases. The patient should be put to bed and kept quiet, or, in less severe cases, confined to the room.

(*b*) *By avoidance of over-action of the heart* by emotional excitement, alcohol, tea, coffee, tobacco, or sexual excesses.

(*c*) *By diminishing peripheral arterial resistance*, should any exist, by regulating the diet, by increasing elimination of offending waste products by the skin, kidneys, and bowels, and by the administration of potassium iodide, chloral, or nitroglycerin.

2. *To improve the force of the heart.* The best drug for this purpose is digitalis. Digitalis is contraindicated in

perfectly-balanced compensatory hypertrophy. The indication for its use is broken compensation, no matter from what valvular affection. When digitalis does good the pulse becomes fuller, more regular, and of better tension, the dyspnœa and œdema diminish, and the urine usually increases in quantity. There are cases in which it does good even if the pulse continues irregular. Toxic effects may, however, be produced by its injudicious administration, and are shown by nausea and vomiting. The urine is reduced in amount. The pulse becomes irregular, and there may be two heart-beats to one of the pulse, especially in mitral stenosis. The particular preparation of digitalis to be used is of no consequence if the drug be good. Only as large doses should be given as may be required; over-stimulation should be avoided. Some patients in serious conditions may require large doses—from 15 to 20 minims of the tincture every three hours—while other cases, less aggravated, do well on from 3 to 4 minims two or three times a day. As a certain increase in the rapidity of the heart is one of the methods of compensation for a valvular lesion, digitalis should not be given blindly to reduce the frequency of the pulse to normal. The proper administration of digitalis requires the greatest judgment and the detailed watching of the patient.

In aortic regurgitation digitalis may do harm by unduly prolonging the diastole, so giving time for the ventricle to become over-distended. In such cases opium in gr. j doses three times a day is often of service.

Strophanthus in the form of the tincture (gr. v to viii) may be employed instead of digitalis. It is often of service in steadying an intermittently acting heart, but it is inferior to digitalis in power.

Convallaria, caffeine, and adonis vernalis are not now so extensively used as formerly. They may be given, however, should digitalis disagree with the stomach. Iron and strychnine are often of great value.

The timely administration of iron, with or without arsenic, often restores tone to the system and checks failing compensation. Strychnine is of great service, combined with digitalis, in increasing the force of the heart. When the pulse is intermittent and irregular, iodide of potassium may be given, either alone or with digitalis. It is often of the utmost service. Nitroglycerin is a valuable heart tonic to meet temporary indications. It may be combined with digitalis.

When the heart's action is rapid and tumultuous, much good is done by cold applications over the heart. When the heart-action is weak and irregular, constant irritation over the heart by a nitric-acid issue is of great service.

3. *To diminish the venous congestions.*

(*a*) *By venesection.* In cases of dilatation from whatever cause, with venous congestions, cyanosis, and dyspnœa, much relief is experienced by the withdrawal of from 15 to 25 ounces of blood. Timely venesection may save the patient's life in acute cases.

(*b*) *By purgation.* This is of service especially in cases with dropsy. From ½ ounce to 1½ ounces of Epsom salt may be given in a concentrated form half an hour before breakfast. The compound jalap powder, or elaterium, or any other hydrogogue cathartic may be given, and is usually well borne.

(*c*) *By diuresis.* For this purpose digitalis, with or without a saline diuretic, potassium citrate or acetate, is most efficient. In almost every case a sure indication that digitalis is doing good is the increase in the quantity of the urine.

Calomel in gr. iij doses every six hours for three or four days is often of the greatest service in cardiac dropsy, acting both as a diuretic and a cathartic. It should be discontinued should stomatitis develop.

A favorite combination is the pill composed of a grain each of powdered digitalis, squills, and blue mass. Iodide of potassium in gr. x doses is often an efficient diuretic. If the blood-tension be abnormally high, diuresis may be increased by the reduction of the tension by iodide of potassium, nitroglycerin, or chloral hydrate. When the urine is greatly diminished, cups and poultices over the kidneys often prove of the utmost value.

(*d*) *By operative interference.* Serous accumulations in the pleural or peritoneal cavities may interfere to such an extent with the respiration and the heart's action that aspiration under the strictest antiseptic precautions may be resorted to. Frequently, after tapping ascitic fluid, diuretics and cathartics, formerly of no avail, will succeed in preventing reaccumulation.

If the œdema of the legs be unrelieved by depletion through the bowels and kidneys or by elevation and bandaging of the feet and legs, scarification of the skin may be resorted to, or Southey's tubes—small silver cannulæ with tubing attached—may be inserted under the skin; but these methods are recommended only in the very severest cases.

4. *To improve the general condition.* While the symptoms incident to the deranged circulation are being treated, every attempt should be made to support the general health of the patient and to control all symptoms that interfere with sleep or with general nutrition. The diet should be simple, nutritious, and easily digestible. Over-distention of the stomach by food or by gas should be avoided. Iron and general tonics should be given. It is important that the patient should enjoy a restful sleep at night. For this purpose sulphonal, chloralamide, or trianol may be given. In milder cases Hoffmann's anodyne, camphor-water, valerian,

or bromide of sodium may suffice. In aggravated cases of insomnia with dyspnœa and restlessness nothing acts more pleasantly than morphine, preferably given hypodermically. Opium by the mouth may be given to these patients in divided doses throughout the day.

3. DISEASES OF THE MYOCARDIUM.

ACUTE MYOCARDITIS.

This disease occurs in two forms:

1. *Acute Diffuse Myocarditis.*—This disease occurs in the course of infectious diseases and in septic processes of all kinds, and seems to be due to poisoning of the heart-muscle by bacterial products. It is best seen in fatal cases of diphtheria. It may complicate endocarditis or pericarditis. The heart-muscle is soft; its color is dark red with hemorrhagic points, or it may be yellowish-red or mottled. The heart-cavities are frequently dilated. The muscle-fibres undergo granular degeneration and may become fatty. The interstitial fibrous structure is infiltrated with round cells. The left ventricle is more frequently involved than the right. The disease may terminate in complete recovery or in chronic fibroid myocarditis, or it may end in suppuration.

2. *Acute Circumscribed Myocarditis, or Acute Suppurative Myocarditis.*—This form of myocarditis is due to infection of the heart-muscle by suppurative micro-organisms which arise from a primary focus of suppuration and reach the heart as emboli. Examination reveals small scattered foci of suppuration in the heart, in the form of grayish or of yellow spots or streaks, usually surrounded by a hemorrhagic zone. They are most common in the anterior wall

of the left ventricle and in the septum, but they may occur in any locality. A suppurative focus may rupture into the pericardium, producing suppurative pericarditis, or into the heart-cavities, producing malignant endocarditis, general septicæmia, or suppurative emboli. These complications may occur without apparent rupture. Aneurysm of the heart and rupture of its wall may occur. The disease is almost always fatal, as the result of the cardiac condition or of the primary disease. Rarely the abscesses become encapsulated, the pus becomes inspissated, or a calcareous nodule may remain.

Symptoms.—The symptoms of both forms of myocarditis are indefinite and are obscured by those of the primary disease. There is, however, a sudden increase of cardiac weakness; the pulse becomes rapid, irregular, and feeble; dyspnœa becomes marked. The heart-cavities usually dilate, and may occasion grave disturbances of circulation. Sudden death may occur, even in patients who have not been considered seriously ill. This termination is especially seen in diphtheria.

The **physical signs** are those of weakened action of the heart, and possibly some increase in its size by dilatation. There may be the murmur of mitral regurgitation from relative insufficiency. The sounds are weak and may be equidistant, giving "tick-tack" sounds resembling those of the fetal heart. The occurrence of such "embryocardia" is always of serious import.

The **prognosis** is always grave except in the lighter degrees of the diffuse form.

Treatment.—The patient should be kept in absolute rest. Cold applications to the precordium seem to be of service, and alcohol should be administered freely. Digitalis does not seem to be of much service; if given in large doses it may be the means of rupturing a heart abscess.

CHRONIC MYOCARDITIS.

Etiology and Synonyms.—This disease may follow acute diffuse myocarditis or areas of anæmic necrosis. It is seen in chronic poisoning by alcohol, syphilis, or gout. It may be associated with pericarditis or endocarditis. Its most common cause, however, is the narrowing of the coronary arteries, producing either low-grade tissue-changes from defective blood-supply, or thrombus-formation resulting in infarctions which gradually become converted to fibroid areas. The affection is thus commonly met with in people of advanced age who have indulged freely in alcohol and high living, who have had syphilis, and who have done hard work. In such patients renal disease, endarteritis, and fibroid myocarditis are usually associated. *Synonyms:* Fibroid heart; Fibroid myocarditis; Chronic interstitial myocarditis.

Pathology.—The lesion may be diffuse or circumscribed. The parts most frequently affected are the wall of the left ventricle, the papillary muscles, and the septum. The affected areas are firm, cut with resistance, and are opaque and grayish in appearance. The lesion consists in the increase of connective tissue with atrophy or degeneration of the muscle-fibres. The coronary arteries generally show obliterating endarteritis. The heart is usually enlarged and hypertrophied. Localized fibroid areas may allow of sacculated dilatation of the heart.

Symptoms.—In some cases of chronic myocarditis there are no symptoms, the lesion being accidentally found post-mortem. In other cases there may be sudden death, which may occur without previous symptoms of disease. In still other cases there are symptoms of weakened power of the heart with circulatory disturbances. Palpitation and dyspnœa are common. There may be attacks of angina pectoris, which may be the only symptom. Intermissions and inequalities of the pulse are common, and the pulse is usually slow, being frequently reduced to 40 or 50 beats in the minute. There may be sudden syncope, coming usually after exertion, in which attack the patient may die. Attacks of coma resembling cerebral hemorrhage may occur and may prove fatal. There may finally be any of the symptoms of a dilated heart with venous congestions.

The **physical signs** are uncertain. The heart is usually enlarged in size; its sounds are weak.

Diagnosis.—Aid is afforded by the presence of the arterial degenerative changes of nephritis and by the occurrence of symptoms of a failing heart without the signs of actual dilatation or valvular lesion. The diagnosis from fatty degeneration is almost impossible.

Prognosis.—The patient may live for years, but death may occur at any time from an attack of angina, of syncope, of coma, or without antecedent symptoms.

Treatment.—In general terms, the treatment is that of fatty degeneration. Iodide of potassium is indicated in syphilitic cases. Heart stimulants are demanded by signs of cardiac weakness. Alcohol, strychnine, and nitroglycerin are to be employed for this purpose, as digitalis is contraindicated because of the already increased blood-pressure in the sclerotic arteries. The diet should be simple; the habits of life are to be well regulated, and exercise is to be taken regularly and sparingly, and never suddenly nor to excess.

SYPHILITIC MYOCARDITIS.

Two forms of syphilitic myocarditis are recognized:

1. *A Diffuse Fibroid Myocarditis.*—This variety cannot be distinguished from the ordinary form by either physical signs or clinical symptoms.

2. *Gummata in the Myocardium.*—These gummatous tumors weaken the heart-muscle, causing symptoms of heart weakness, and may result in sudden death or in rupture of the heart. A positive diagnosis can be made only in cases where marked improvement follows the administration of large doses of potassium iodide in syphilitic subjects with heart weakness.

DEGENERATION OF THE MYOCARDIUM.

1. Anæmic Necrosis, or white infarct, a localized degeneration of the myocardium, occurs as the result of the occlusion of a coronary artery or of one of its branches by thrombosis or embolism. Thrombosis is favored by sclerosis and atheroma of the wall of the vessel. The anterior coronary artery is usually the one involved, so that the resulting anæmia-necrosis is found in the left ventricle or in the septum. The patch is anæmic, whitish or grayish in color, and is usually of an irregular wedge shape. It may soften and break down, and may even result in rupture of the heart, or it may undergo hyaline degeneration and ultimately become sclerotic, forming the lesion of fibroid myocarditis. Such a blocking of the coronary artery is one of the common causes of sudden death, and the lesion should always be looked for in these medico-legal cases. In other cases there are angina pains with feebleness of the heart's action. There may be a series of such attacks, any one of which may prove fatal.

2. Parenchymatous degeneration, or "cloudy swelling," is seen in the course of infectious diseases, especially diphtheria, typhoid, and scarlet fever. It may occur even if the temperature be but slightly elevated. The left ventricle is most markedly involved: its walls are pale, turbid, and exceedingly soft and flabby; its cavity is usually somewhat dilated. The muscle-fibres are seen filled with numerous

fine granules obscuring the striæ and the nuclei. There may be some infiltration of the interstitial connective tissue with round cells, and the nuclei of the muscle-cells are usually swollen and multiplied. The degeneration may merge into fatty degeneration. The symptoms are those of acute diffuse myocarditis, and a differentiation from that disease by clinical symptoms and physical signs is impossible.

3. FATTY HEART.—The term "fatty heart" is loosely applied to either of two distinct conditions, fatty infiltration and fatty degeneration.

(1) *Fatty Infiltration* (*Cor Adiposum*).—In general obesity the normal amount of fat covering the heart is much increased; bands of fatty tissue may extend between the muscular fibres even to the endocardium and the papillary muscles. The muscular fibres may be normal or atrophied, or they may undergo pressure-degeneration. The heart-wall is weakened and may dilate or rupture. Such fatty infiltration occurs with general obesity, usually between the fortieth and seventieth years, and is more common in men than in women. It may more rarely be seen in the conditions of old age and cachexia.

The *symptoms* are indefinite. There is dyspnœa on exertion, due either to the general obesity or to enfeebled heart-power. The pulse is usually weak and rapid. There may be angina pains. Sudden death may occur from rupture of the heart.

The *physical signs* are elicited with difficulty because of the increased thickness of the chest-wall. The area of cardiac dulness may be increased by the fatty deposit. The heart-sounds are weak; there may be a systolic murmur at the apex, from relative insufficiency of the mitral valve.

The *treatment* is that of general obesity, by regulated diet and systematic physical exercise.

(2) *Fatty Degeneration.*—*Etiology.*—The heart is very

subject to this form of degeneration. By reason of its incessant activity the heart needs an abundant supply of oxygen, and it is the most susceptible muscle in the body to show changes in nutrition. Any cause, therefore, preventing an abundant supply of good blood, or preventing good circulation of blood within the heart itself, will be followed by degeneration. Moreover, the heart-muscle is most susceptible to bacterial and chemical poisons.

(*a*) *Failure of general nutrition* in old age, in cachectic states, and in wasting diseases. Fatty degeneration occurs in acute and chronic anæmia, and is more common in those who lead a sedentary life.

(*b*) *Failure in local nutrition.* Fatty degeneration complicates chronic pericarditis with adhesions; dilatation of the heart, or heart weakness from any cause by which the circulation of blood in the coronary arteries is allowed to become sluggish by reason of feeble contractile power of the ventricular wall; aortic regurgitation, in which the diminished arterial tension does not allow of efficient filling of the coronary arteries. It may occur in the hypertrophied heart of valvular disease. It is common with disease of the coronary arteries. As this latter condition is usually secondary to atheroma of the aorta, fatty degeneration of the heart should always be suspected in old people with atheromatous changes of the aorta and the aortic valves associated with a weakly-acting heart.

(*c*) *Poisoning of the heart-muscle.* This complication may occur with severe infectious disease, especially diphtheria and typhoid fever, and may be associated with fatty degenerations in other viscera. It is seen in an intense form after poisoning by phosphorus or by arsenic. It may be caused by long-continued intemperance, or it may occur with diabetes.

Fatty degeneration occurs more frequently in men than in women, and is usually a disease of adult life or of old age. In some cases no assignable cause can be found.

Pathology.—The process may be general or local. The left ventricle is usually, however, affected. At first there appear yellowish striæ and points under the endocardium, especially in the papillary muscles and the trabeculæ, the remainder of the myocardium being healthy. In more marked cases, such as are seen in profound anæmia, the entire heart may be of a light-yellowish color and be very feeble and flabby, the heart-muscle often tearing easily. There may be areas of a brownish color—the so-called "brown atrophy." This is especially seen in cases associated with valvular disease or senility. The heart-cavities may be dilated, and in extreme cases may rupture. Microscopically, the muscular fibres are seen to be filled with fatty granules and oil-drops; the striæ and the nuclei are indistinct. In severe cases the fibres seem completely occupied by the granules. The areas of brown atrophy, when present, show the color to be due to a deposit of yellowish-brown pigment about the nuclei.

Symptoms.—In some cases sudden death occurs, with or without previous indications of cardiac trouble. Such a fatal event may follow the giving of ether or chloroform, sudden mental shocks or emotions, after exertion, or after a hearty meal. In other cases there are more definite symptoms.

Usually symptoms of cardiac insufficiency appear. There may be dyspnœa on exertion, or it may be constant. The pulse is short and unsustained. Such a pulse may be a constitutional peculiarity in some persons, but is suspicious if met with in old people. The pulse may be regular or irregular, frequent or slow, falling at times even to 8 or 10 to the minute. A slow pulse of low tension is characteristic when it occurs, but it is rather rare. The pulse may become rapid and irregular, and may "go to pieces" upon exertion, whereas the pulse of functional disease of the heart becomes stronger and more regular on exertion.

Extreme fatty changes, however, may be consistent with a full regular pulse and regular heart's action, provided dilatation of the heart does not occur. It seems, then, that the symptoms really depend upon the supervening dilatation. When dilatation occurs, there are the ordinary symptoms of such condition. It can usually, however, be differentiated from dilatation from other causes by attention to the following points: (1) The heart is not always much increased in size. (2) The symptoms are more constant. (3) Œdema and anasarca are exceedingly uncommon. (4) There are frequently present symptoms of a certain diagnostic value. They comprise symptoms of syncope, pseudo-apoplectic and epileptic seizures, and angina pectoris.

The *syncopal attacks* are characterized rather by their duration than by their intensity. There is never entire loss of consciousness, but the attack may continue with feeble heart-action, frequently a pulse sinking to 30 or 40 to the minute, and cold, clammy skin for hours. Such an attack in an old person or in one in whom a sufficient cause for fatty degeneration is present is exceedingly significant.

The *pseudo-apoplectic* attacks are characterized by the sudden onset of coma with stertorous breathing, often of the Cheyne-Stokes variety. There may even be a temporary hemiplegia. Absence of raised arterial tension and of the characteristic temperature-curve differentiates this condition from cerebral hemorrhage. The attack is probably due to circulatory disturbances of the brain from a weakly-acting heart. From such an attack the patient may recover, but he is always mentally enfeebled.

The *epileptiform* attacks resemble those of *petit mal.* There are convulsive movements, which are not usually severe. The patient is partially unconscious, not as in epilepsy, but more as in syncope. The pulse is usually slow, often as low as 20, and of low tension. There may be mental delusions or mania following such an attack.

The *angina* attacks are identical with those of the true or the false angina.

Physical Signs.—There are no essential physical signs in fatty degeneration. There need be no increase in the size of the heart, and no murmur unless from pre-existing valvular disease. There may, however, be evidences of dilatation consequent upon the fatty degeneration, and a systolic murmur at the apex, due to relative mitral insufficiency. The heart's impulse is weak, vibratory, or absent. The first sound is short—a suspicious sign in old people; the sounds may be equidistant, and the gallop rhythm may be present. The absence of physical signs adequate to explain the symptoms of cardiac inefficiency is of the greatest aid in diagnosis.

Prognosis.—Mild cases following anæmia, wasting diseases, and fevers usually do well. The symptoms are never well marked, consisting usually only of a rapid weak pulse and some little dyspnœa on exertion, with a tendency to syncopal attacks. The heart-muscle returns to a state of health when the general health of the patient improves.

The prognosis of the severer forms is bad. Fatty degeneration occurring in a hypertrophied heart with valvular disease weakens the muscular wall, allows of dilatation, and upsets compensation. Sudden death may occur at any time, either unexpectedly, as during an attack of syncope, pseudo-apoplexy, epilepsy, or angina, or from rupture of the heart. The patient may die from heart weakness should he be attacked with any intercurrent disease.

Treatment.—The patient should avoid every physical or mental excitement that might tax the power of the heart.

Rest is of the utmost importance. The diet should be simple and nourishing. The stomach should not be overdistended by food or by gas. The strictest attention is to be paid to the general health. Anæmia should be met with iron tonics; malnutrition, by proper feeding, cod-liver oil, and fresh air. Wine with the meals may be allowed, to stimulate digestion.

Symptoms of heart feebleness should be controlled by heart stimulants and strict enforcement of rest. Digitalis is often of great service, but any of the other cardiac tonics may be employed. Nitroglycerin is to be employed if the tension of the pulse be high from associated arteriosclerosis. The general management of the case is that of valvular disease with broken compensation. Angina attacks are best relieved by amyl nitrite, nitroglycerin, or morphine given subcutaneously, while sudden attacks of heart failure require active stimulation by inhalations of amyl nitrite or ammonia or by hypodermics of ether, whiskey, or digitalis.

ANEURYSM OF THE HEART.

1. *Aneurysm of a valve* results from weakening of the valve by either simple or malignant endocarditis. Aneurysms of the aortic valve bulge into the left ventricle; those of the mitral valve, into the auricle. The aortic valves are most frequently affected, the anterior mitral segment being more often involved than the posterior. Rupture of a valve-aneurysm produces extensive destruction and incompetency.

2. *Aneurysm of the heart-wall* is preceded by weakening of the wall by chronic myocarditis, by endocardial ulcerations of malignant endocarditis, and by areas of anæmic necrosis. In rare cases aneurysm of the heart-wall has followed stab-wounds. The usual situation of an aneurysm is in the left

ventricle near the apex. Aneurysm of the auricles or of the right ventricle is rare. The aneurysm may vary in size from that of a nut to that of the heart itself. Its sac is composed of pericardium, myocardium (the muscular fibres of which are often replaced by fibrous tissue), and endocardium. The cavity of the aneurysm is frequently occupied by laminated fibrin. Rupture of the aneurysm has occurred in but 7 out of 90 cases.

The **symptoms** are not distinctive, and a diagnosis is rarely made. There may be near the apex some localized bulging, which may give an expansile pulsation. If the aneurysm be large, there may be marked disproportion between its pulsation and the feeble pulsation in the peripheral arteries.

The **prognosis** is exceedingly grave. Death may result from syncope or rupture, but more usually it occurs gradually from heart exhaustion due to the primary disease.

The **treatment** is that of fatty heart. Nothing can be done directly for the aneurysm.

RUPTURE OF THE HEART.

Etiology.—A degenerated condition of the myocardium must in all cases precede rupture of the heart. Fatty degeneration, especially of localized areas, is the most frequent cause, occurring in 77 per cent. of all cases, but anæmic necrosis following thrombosis of the coronary arteries, fatty infiltration, circumscribed myocarditis, broken-down tumors and gummata, and deep endocardial ulcerations and cardiac aneurysms may also lead to rupture. Two-thirds of all subjects of rupture of the heart are over sixty years of age. The rupture usually occurs after exertion, but it may occur while the patient is at rest.

Pathology.—The usual situation of the rupture is in the anterior wall of the left ventricle, near the apex; more rarely the rupture may be situated in the posterior wall of the left ventricle, in the septum, or in the wall of the right ventricle. The rupture is usually small, and it may be either direct or indirect.

Symptoms.—If the rupture be direct, the patient experiences agonizing cardiac pain, suffocation, and great apprehension. The pulse becomes rapid and feeble; the skin is cold and clammy. Death may occur in syncope in a few minutes (in 71 per cent. of cases), or it may be deferred for several hours. In the more protracted cases vomiting and purging may be noticed.

The **prognosis** is always fatal.

The **treatment** is entirely prophylactic. Persons known to have degeneration of the myocardium should lead tranquil lives free from every mental or bodily strain.

4. NEUROSES OF THE HEART.

PALPITATION.

The term "palpitation" is applied to all forms of abnormal cardiac sensations which are unpleasantly sensible to the patient. The destructive features are violent pulsations of an unpleasant nature, usually with throbbing of the larger arteries. The pulse may be rapid and over-forcible, but it may be normal or even weak. The attack appears suddenly, lasts a few minutes or hours, and, while not serious, occasions considerable alarm. Various neurotic symptoms—flushing of the face, sweating, eructation of gas, and the abundant passage of limpid urine—often accompany or follow the attack.

Palpitation is a pure neurosis, which may, of course, occur in a healthy or a diseased heart. It is to be distinguished from the over-action of organic disease in that it is not produced by exertion—in fact, is often dispelled by exercise—appears often at night while at rest, and is not accompanied by dyspnœa or other symptoms of cardiac distress.

Pathology.—There is no lesion, but in long-continued cases hypertrophy or dilatation may result.

Etiology.—The cause of palpitation of the heart is a reflex inhibition of the vagus action that enables the accelerators to run away with the heart. The affection is common in women and in young adults, and is rarer in advanced age. It occurs in weak and nervous conditions, after sickness, in hysteria and neurasthenia, in excitable subjects, and at the climacteric. It is produced by unhealthy occupations and by vicious modes of life. It is common with over-use of tea, tobacco, or coffee. Flatulent dyspepsia is a prolific cause of palpitation, and it may occur as a reflex phenomenon from gastric, intestinal, or ovarian irritation. The effect of emotions is well known. Palpitation of the heart is a symptom of exophthalmic goitre.

The "irritable heart of soldiers" (DaCosta) is a form of palpitation caused by excitement and over-exertion, especially if the thorax be compressed with shoulder-straps; this condition is accompanied with some dyspnœa on exertion.

TREMOR CORDIS.

This condition, which is the opposite of palpitation, occurs occasionally in youth and more commonly in advanced life. It may occur in healthy hearts or in those enfeebled by myocardial degeneration. The attack comes without warning; the heart "trembles" or "flutters," while the pulse sinks to a tremulous thread. The attack lasts for a few seconds and terminates by a forcible cardiac beat.

Tremor cordis is almost always due to flatulence or cardiac distress, and is not produced by emotions. There is no accompanying faintness, although the attack occasions serious alarm.

The **prognosis** is perfectly good.

INTERMITTENT ACTION.

Intermittent action occurs whenever the heart misses a beat from time to time. Intermittency may be regular or irregular, habitual and constant or only occasional. It usually occurs after meals, as an evidence of flatulent dyspepsia; it occurs after over-use of tea, coffee, or tobacco; it is common in gout, in uric-acid diathesis, in nervous and hypochondriacal conditions, and after bodily and mental shocks. A constantly intermittent action of the heart is common to many old people, and is of no serious significance.

Intermittency often occurs with fatty degeneration of the heart, and is to be distinguished from reflex intermittency by getting the patient to exercise briskly. By such exercise the really weak heart goes to pieces, while the healthy but neurotic heart clears up.

Intermittency associated with organic disease of the heart is often of serious omen, indicating that the contraction of the auricles is not sufficient to fill the ventricles, hence the ventricles wait until they are properly filled.

TACHYCARDIA.

A rapid pulse accompanies many morbid conditions, such as fevers, exhaustion, collapse, emotional conditions producing palpitation, pain, maniacal conditions, and the ingestion of certain poisons, as alcohol, atropine, nitroglycerin, and over-doses of digitalis. A rapid pulse is physiological after exertion and in the newly-born, whose normal pulse ranges between 120 and 135.

The term "tachycardia," or "heart-hurry," is more properly applied to a rapid heart-action, often reaching 200 or more in the minute, the action being, moreover, usually feeble. Its distinguishing feature is the very little disturbance it gives, in contradistinction to the rapid pulse of exophthalmic goitre, of palpitation, of exertion, and of cardiac failure. Tachycardia may be due to tumors pressing on the vagus trunk or to mitral stenosis. In later life it is an important sign of senile degeneration, and is an added source of danger, as the attack may terminate in syncope or in asystole.

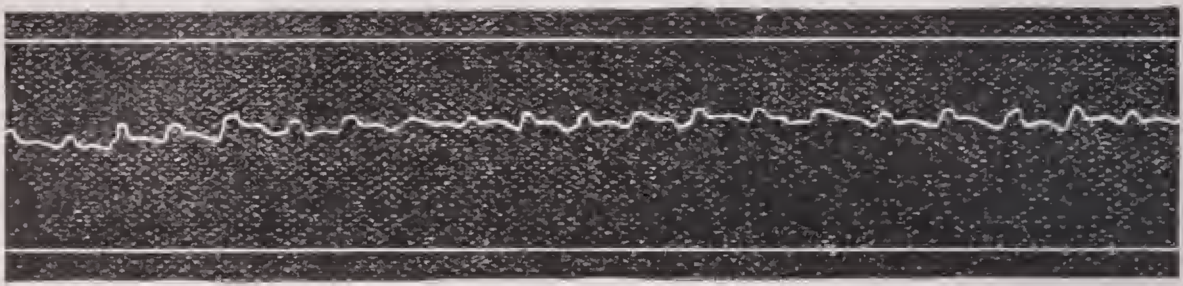


FIG. 18.—Sphygmogram from a case of tachycardia. Pulse-rate, 175.

Reflex tachycardia, a pure neurosis, may occur from any source of irritation, especially from gastric distress, and in reflex tachycardia the pulse may be fairly forcible.

Intermittent tachycardia is a rare disorder in which heart-hurry comes in attacks at varying intervals, each attack lasting a few hours. The pulse is rapid and weak, frequently over 200 to the minute, but usually the distress to the patient is but slight. The cause for such attacks is not definitely known. A permanent cure is rare, and the disease may terminate fatally at any time.

BRACHYCARDIA; BRADYCARDIA.

Two forms of slow heart are recognized—the false and the true.

False brachycardia, in which the pulse is slow but the number of heart-beats is normal, is usually due to a dilating heart with myocardial degeneration, in which the dropped beats are due to weak or abortive systole. In some cases false brachycardia seems to be due to alternating hemisystoles, each ventricle acting independently.

True brachycardia occurs whenever both pulse and cardiac systole are abnormally infrequent.

Infrequent pulse occurs in some people as a constitutional peculiarity; it occurs in hunger, and it is frequent in the puerperal state. Pathologically it occurs in a number of conditions:

(1) In diseases of the medulla and in compression of the brain; for example, in basilar meningitis, tumors of the brain, and cerebral hemorrhage. It occurs also with diseases and injuries of the cervical cord.

(2) In degeneration of the heart-muscle. A slow pulse in the aged is highly significant of fatty heart. The pulse may fall to even 8 in the minute in these cases.

(3) After the sudden lowering of peripheral resistance, as by bleeding or by the withdrawal of effusions in the chest or the abdomen.

(4) After the critical fall of temperature in acute fevers, especially in pneumonia, typhoid fever, erysipelas, and acute articular rheumatism.

(5) In poisoning by digitalis, alcohol, aconite, or lead. Infrequent pulse occurs regularly with jaundice. It is common in uræmia. The poison may be from auto-intoxication following digestive disturbances—a very common cause for brachycardia. Thus it is frequently observed with ulcer, cancer, or dilatation of the stomach.

(6) Conditions of asphyxia.

(7) Various conditions of melancholia.

Brachycardia is most common with advanced life. The affection is usually of serious import, as in senile hearts it is very often dependent upon dilatation and myocardial degeneration.

The **treatment** is that of the underlying cause.

ANGINA PECTORIS.

Definition and Synonyms.—Angina pectoris is a disease characterized by severe pain over the heart, by a sense of impending death, and in severe cases by disturbances in the action of the heart. *Synonyms:* Stenocardia; Breast-pang; Neuralgia of the heart.

Etiology.—The disease, which is one of adult life, and usually of the higher classes, attacks men in over 80 per cent. of all the cases. The exciting cause of an attack may be either exertion, external cold, indigestion, or constipation, subsequent attacks becoming more and more easily provoked. In some cases no exciting cause çan be discovered.

Lesion.—No one lesion is constant. Ossification or inflammation of the coronary arteries is commonly found. Fatty heart, arterial sclerosis leading to high arterial tension, atheroma of the aorta or of the aortic valves, fibroid myocarditis, and chronic inflammation of the coronary plexus are also among the pathological findings. In some rare cases no lesion is discoverable.

The nature of the disease is not well known, The best explanation seems to be that a sudden high tension occurs in the arteries, causing a spasm of the heart in its efforts to overcome the resistance. The same spasm occurs in an over-filled bladder or stomach when its contents cannot easily be expelled, causing often agonizing pain. As a matter of fact, extreme degrees of arterial tension occur in 96 per cent. of all cases of angina pectoris during the attack. In the 4 per cent. of cases in which the tension is not raised no explanation can be given. There being in almost all cases some form of cardiac lesion, angina pectoris affords the strongest presumptive proof of organic disease.

Symptoms.—The symptoms occur in attacks lasting from a few seconds to half an hour. One or two minutes is the average duration. The severer attacks occur at night. Each attack is attended by three cardinal symptoms:

(1) *Pain* over the heart is exceedingly severe; it is neuralgic in character, with a sense of constriction. The pains may radiate up the neck and down the left arm and hand, or they may extend to the back. The face is cold, ashen-pale, and clammy; the expression is anxious; there may be general sweating. The patient immobilizes himself from pain. With the pain there may be a feeling of numbness or coldness in the fingers.

(2) *Dread of impending death* coexists with the pain in an equal degree. After the attack is over the patient frequently exclaims that if the pain had lasted a minute longer he would have died. In attacks cut short by the use of drugs the sense of impending death may not be noted. In mild degrees the patient may complain only of uneasiness and general apprehension.

(3) *Disturbance in the heart's action* occurs in almost all attacks. The pulse becomes rapid and irregular, and in 96 per cent. of cases is of extremely high tension. In some cases, however, the pulse may be uniform and but slightly altered.

There may be considerable dyspnœa, and during the attack there may be a vehement desire to pass urine, although the bladder be empty.

The attack may terminate in recovery, usually with the eructation of gas, with vomiting, or with the passage of a large quantity of limpid urine; or the patient may pass into syncope, from which he may or may not recover.

Some patients who have had angina suffer from time to time from attacks of faintness without either pain or dread. This condition is not really angina, but is equally as serious.

There may be but one attack of angina, or there may be a number of attacks at irregular intervals of weeks, months, or years.

Between the attacks the disease itself gives no symptoms, although the underlying cardiac lesions present their ordinary clinical symptoms and physical signs.

The **prognosis** is bad. The patient may die in many of the attacks, some patients not surviving the very first. Recurring attacks, as a rule, become more frequent and more severe, although it is possible for the attacks at any time to cease recurring. Much can be done by judicious treatment. The prognosis is best in cases in which the attacks are induced by a preventable cause. The disease often runs a protracted course, and is not so serious if associated with aortic disease.

Treatment.—*During an Attack.*—Treatment is directed toward the blood-tension and the pain. If the arterial tension be increased, amyl nitrite should at once be given by inhalation, from 2 to 5 drops being placed in cotton or on a handkerchief and applied to the nose. Patients subject to attacks of angina pectoris should carry with them constantly the pearls of amyl nitrite, and should use them at the first indication of an attack. Usually this treatment cuts short an attack, but it may fail. If relief is not afforded in a minute or two by amyl nitrite, chloroform should be given, a few inhalations often affording prompt relief. In some cases a hypodermic injection of morphine must be resorted to; its action is rendered more efficient by a hot bath. In case the arterial tension be not increased, amyl nitrite does not do much good, and the treatment consists chiefly in morphine combined with inhalations of chloroform.

Between the Attacks.—The general health should be cared for in every possible way. Excitement and sudden or severe muscular effort should be avoided. Tobacco and stimulants are prohibited. Exciting causes of attacks should be found and prevented. If the blood-tension is high between the attacks, it should be reduced by regulation of the diet and the action of the kidneys and the bowels and by the use of drugs. For this purpose nitroglycerin may be given in gr. $\frac{1}{100}$ doses, at first three times a day, the dose being increased gradually until the patient complains of flushing or of headache.

Prolonged use of iodide of potassium is often followed by good results. From 10 to 20 grains, three times a day, may be given for years, the dose being omitted from time to time or being replaced by doses of nitroglycerin to avoid iodism. There may be good results obtained by combining chloral hydrate in 5- or 7-grain doses with the iodide.

PSEUDO-ANGINA.

Etiology.—Women are more frequently affected with pseudo-angina than are men. The disease is most common in the nervous and neurasthenic, and is apt to be associated with other nervous and vaso-motor phenomena. It may occur at any age. It is common at the menopause, and may occur with especial frequency at the monthly sickness. The attacks may arise spontaneously, or they may be precipitated by worry or by disturbing emotions. In some cases there is a distant reflex origin.

Pathology.—Pseudo-angina is a pure neurosis, and there is no essential lesion.

Symptoms.—The disease comes in attacks which last for minutes, days, or even for weeks. If the attack be prolonged the symptoms are remittent. The average duration is one or two hours, being longer than in true angina. The attacks, which may recur with a certain periodicity, are usually more frequent than in true angina. The symptoms of an attack somewhat resemble those of true angina. There is pain over the heart, which, however, is less severe, more diffused, and often is accompanied with precordial tenderness. The patient does not immobilize himself as in angina, but is agitated and anxious. There is not the same dread of impending death, although the patient is apprehensive. The heart's action is either feeble and irregular or tumultuous. The arterial tension is not increased. The breathing is rapid and oppressed. Vomiting and pain over the stomach are common toward the close of protracted attacks. There may be various hysterical or neurotic symptoms during and between the attacks.

The **diagnosis** is chiefly to be made from true angina. The chief points of diagnosis have already been given. The absence of arterial or cardiac lesions would be of importance in excluding the true angina. There are, however, difficult cases of combined hysteria, aortic valvular disease, and angina pains in young women, in which an absolute diagnosis cannot be made.

The **prognosis** is perfectly good both for life and for recovery.

Treatment.—During the attack the action of the heart should be regulated; if the attack be prolonged, sedatives and anti-neuralgic remedies may be employed, such as phenacetine, bromide of sodium, and camphor or cannabis indica. As the arterial tension is not increased, amyl nitrite, nitroglycerin, and similarly acting drugs are not indicated. Between the attacks treatment should be directed toward the general health and the underlying nervous condition.

EXOPHTHALMIC GOITRE.

Definition and Synonyms.—Exophthalmic goitre is a disease characterized by enlargement of the thyroid gland, protrusion of the eyeballs, and tachycardia, together with various nervous phenomena. The disease was first described in 1786 by Parry, but a complete description was first given in 1835 by Graves of Dublin, and in 1840 by Basedow of Germany. *Synonyms:* Graves' disease; Basedow's disease.

Etiology.—While no age is exempt, exophthalmic goitre is most common between the ages of fifteen and thirty-five. It occurs in women in the proportion of 5 to 1. When it occurs in men it seems to run a more severe course. It is most common in anæmic nervous people, and may run in families predisposed to nervous ailments. It may follow a blow, a shock, or a fall. It may occur after pregnancy, although when pregnancy occurs in a patient affected by the disease, recovery more or less complete may ensue.

Pathology.—The nature of the disease is unknown. A supposed lesion has been sought for in the sympathetic nervous system, but changes in the nerves and the ganglia are neither constant nor peculiar. The disease can be reproduced in dogs by destruction of the restiform bodies, and a case has been reported in which hemorrhages were found in the floor of the fourth ventricle. No one theory at present advanced explains all the symptoms of the disease.

The **symptoms** of exophthalmic goitre may be divided into four groups:

1. *Heart Symptoms.*—The heart's action becomes rapid, running frequently as high as 120 to 140, or even 200, beats in the minute. The rapidity is largely controlled by the

conditions of rest and exertion. The action of the heart is usually forceful and accompanied by a feeling of palpitation, but both these latter symptoms may be absent. The tachycardia is usually the first symptom observed, and, in fact, the disease may stop here, with this as its only symptom. There is usually marked pulsation of the carotids, and there may be a capillary pulsation. In long-continued cases there may be hypertrophy of the heart, which may in debilitated subjects merge into dilatation. Soft systolic murmurs at the base are common; they may be heard at the apex as well.

2. *Exophthalmos* usually follows the tachycardia. The eyeball is protruded, and the eyelids do not cover the sclerotics, leaving a rim of white above and below the cornea, giving the patient a peculiar startled look. The protrusion may be extreme, so that the eye is dislocated from its socket. There is a lack of synchronism between the action of the eyeball and that of the upper eyelid, so that when the eyeball is moved downward the lid does not follow it as in health. This is known as "Graefe's symptom." The upper eyelid may be so retracted that it is retained near the bony wall of the orbit, and to this condition is given the name of "Stellwag's symptom." The pupils and the optic nerves are usually healthy, but pulsation of the retinal vessels is common. Exophthalmos may be absent in some cases.

3. *Goitre* develops with the exophthalmos. It may be general or in only one lobe, and the enlargement is rarely so extreme as in simple goitre. The gland is soft and pulsating at first, becoming firmer and harder in protracted cases. There is usually a thrill felt on palpation. On auscultation may be heard a systolic murmur, or more commonly a venous hum.

4. *Nervous symptoms* are common to almost all cases, but there is considerable variety in the extent to which they are developed.

Emotional and mental disturbances are common. There may be hysteria, neurasthenia, irritability of temper, and mental depression often passing into melancholia. There may be temporary mania. There is a tendency to general neuralgic pains. Symptoms of general paresis have been observed in a few instances.

Muscular tremors comprise one of the most constant symptoms of the disease. The tremor is usually fine, generally first involving the hands, and is more marked on motion. It may become general, and may even interfere with the walking power. In rare cases the tremor may be limited to one member.

There is usually insomnia. There may be attacks of precordial pain resembling pseudo-angina. The skin is persistently moist with perspiration, and the electrical resistance of the body is diminished.

Derangements of the digestion are common. There may be intermitting attacks of diarrhœa and flatulency, with severe and distressing vomiting resembling the gastric or gastro-intestinal crises of locomotor ataxia. There may be pigmentation of the skin as in Addison's disease, or patches of leucoderma. The hair may become white or may fall out. Urticaria and angio-neurotic œdema of the skin are common. There may be slight irregular fever without known cause.

Alternating flushings and pallor of the face with hot and cold flashes are common. Menstrual disorders frequently occur, amenorrhœa being the rule, although menorrhagia may occur. There may be paroxysmal dyspnœa occurring especially with the attacks of palpitation.

Prognosis.—Exophthalmic goitre runs usually a chronic course extending over years. It is seldom fatal except from the dilatation of the heart that may be induced. A certain number of patients recover completely or in part, but when the disease is well developed recovery is rare. There are some acute cases following fright in which recovery is rapid.

Treatment.—The general nervous and anæmic condition should receive efficient treatment. The diet should be nutritious and digestible. Moderate exercise in the open air is to be enforced systematically if possible. All causes for nervous irritation should be avoided, and iron with arsenic or inhalations of oxygen gas should be given, together with general nervines and tonics. The action of the heart should be regulated. Digitalis and strophanthus may succeed, but they are not reliable. Convallaria often does good when digitalis fails. Ergot may be employed with benefit, and belladonna given until dryness of the throat is obtained may be of service. Brilliant results often are obtained by iodide of potassium in 5- or 10-grain doses three times a day combined with from 10 to 15 grains of sodium bromide. This combination is especially indicated where the heart's action is rapid and forceful with subjective feelings of palpitation.

Aconite and veratrum viride are not of much benefit. Rest in bed with cold applications (as an ice-bag or Leiter's coil) over the heart are often efficient. In less severe cases a smooth piece of ice may be rubbed briskly over the heart for fifteen minutes two or three times a day.

The use of galvanism has been recommended highly, and it should always be tried as a routine measure. The cathode should be placed at the back of the neck or at the angle of the jaw, while the anode is placed over the course of the sympathetic in the neck or over the heart. An application for fifteen minutes every second day is sufficient.

Feeding with raw thyroid glands of the sheep and hypodermic injections of thyroid extract have been employed, but with unfavorable results. In severe cases thyroidectomy may be resorted to with a fair percentage of cures and improvements; but the operation is in itself a considerable source of danger. Ligation of the thyroid arteries has been recommended.

5. CONGENITAL MALFORMATIONS.

Congenital malformations may be due to arrested or abnormal development or to endocarditis during fetal life. The following classification is the one generally adopted:

Patency of the Foramen Ovale.—If the patency exists in but small degree, it is not of serious importance unless accompanied by other anomalies. The greater degrees of patency are not incompatible with fairly prolonged life. In these cases the diagnosis can be suspected by marked cyanosis, either without heart-murmurs (fairly diagnostic) or by systolic and presystolic murmurs heard over the mid-sternum at the level of the third and fourth ribs.

Defects of the Septa.—Minor defects of the ventricular systole are not rare and are not of much importance. Major defects rarely occur alone, but usually are associated with stenosis of the pulmonary valve, forming a most serious lesion. The defect results in the propulsion of blood from the stronger left ventricle into the right heart during systole, leading to embarrassed respirations and venous congestions. The physical sign is a loud systolic murmur heard over the whole precordium and between the shoulders, and not transmitted. Both auricular and ventricular septa may be defective, producing the *cor biloculare*, or the reptilian heart.

Stenosis or Incompetence of the Tricuspid and Mitral

Valves.—These defects are rare. For physical signs see Chronic Endocarditis, pp. 223–249.

Stenosis and Atresia of the Pulmonary Orifice or of the Conus Arteriosus.—These affections comprise the most important group of cases, being, moreover, relatively common. Pulmonary stenosis alone is not inconsistent with life for some years. For the physical signs see Pulmonary Stenosis, p. 243. The lesion, however, is usually associated with defects of the ventricular septum, and the prognosis is thereby rendered far more serious.

Persistence of the Ductus Arteriosus.—This channel should normally be closed on the fourteenth day. If patent, there result rapid hypertrophy and dilatation of the right ventricle, dilatation of the pulmonary artery, dyspnœa, cyanosis, and congestion of the lungs, with general venous congestions. The physical signs are a long-continued systolic murmur over the pulmonary area, a systolic thrill, and a protrusion of the upper part of the sternum.

Stenosis of the Aortic Orifice.—Stenosis of the aorta is rare. It is a serious lesion, incompatible with life for more than a few weeks. Stenosis of the conus arteriosus is not inconsistent with a fairly prolonged life. The circulation is carried on by anastomoses between branches of the subclavian with those of the epigastric and intercostal arteries. The arteries of the upper extremities are regularly larger and fuller than those of the lower.

Transposition of Arterial Trunks.—This condition is necessarily fatal *in utero*, unless compensated by other anomalies, such as open foramen ovale or communication between the pulmonary vein and the right side of the heart.

Numerical Anomalies of the Valve-segments.—Supernumerary valves are of no significance. Deficiency of valve-segments is usually associated with other and more serious anomalies.

Ectopia cardis may occur with congenital fissure of the sternum and abdomen. Displacement of the heart into the neck or the abdomen may occur. There may be acardia, double heart, bifid apex, or absence of the pericardium.

Symptoms.—Radical defects are inconsistent with life, so that the child dies *in utero* or shortly after birth. In those who live cyanosis is so marked a feature that the name "morbus cæruleus" has been given to the disease, and the term "blue babies" has been applied to these children. The cyanosis, which may be constant or may be induced only by exertion or by crying, varies from a lead color to a purplish hue. The child shows retarded mental and physical development. The nails are clubbed. The external temperature is low, and there is a great susceptibility to cold. Pulmonary affections are common, dyspnœa and cough are frequently observed, and the child is apt to succumb to an attack of bronchitis or of pulmonary congestion, or to any of the ordinary diseases of childhood.

Treatment consists in guarding the child from cold and in checking promptly intercurrent diseases. The treatment for the heart itself is the same as that for valvular disease of adults.

6. DISEASES OF THE ARTERIES.

ARTERIO-SCLEROSIS.

Etiology and Synonyms.—Arterio-sclerosis occurs as a disease of advanced age, usually in those over forty. It is one of the conditions of senility. Predisposition to arterial degeneration runs in some families and may be inherited. Among the exciting causes are chronic alcoholism, lead-poisoning, gout, syphilis, diabetes, and over-eating, especially with sedentary habits of life. There may be an antecedent history of uric-acid diathesis. The disease is common with chronic articular rheumatism, and may precede, follow, or develop simultaneously with Bright's disease, especially with the granular kidney. It may follow severe over-work of the muscles. *Synonyms:* Chronic endarteritis; Atheroma.

Pathology.—The aorta is almost always affected; next in frequency come the larger arteries. The disease may be uniformly distributed, or it may involve some arteries and not others; it may be either circumscribed or diffuse. The intima is much thickened by an increase of connective tissue and by the deposit of round cells. These cells may undergo fatty degeneration, imparting a yellow color in patches to the interior of the artery. In the deeper tissues the cells may break down to form a mixture of fat, detritus, and cholesterin-crystals from which the name "atheroma," or "pulp," is derived; or, if near the surface, an atheromatous ulcer is formed. Atheromatous patches and ulcers and the narrowed lumen allow of the formation of thrombi. In other cases the intima becomes markedly sclerotic and of bony hardness from the deposit in it of salts of lime. The media and the adventitia may show similar changes, even to fatty degeneration and calcification, or the media may become atrophied.

Secondary lesions are found chiefly in the heart. The arterial lesion produces loss of elasticity, and consequently increases peripheral resistance to the work of the heart. The thickening of the intima may, moreover, cause such a narrowing of the lumen of the vessels that an added resistance results; in consequence, the left ventricle becomes hypertrophied in all cases in which the nutrition of the patient is good, and compensation is effected. Should general nutrition fail, dilatation will ensue.

The hypertrophied heart, pumping blood into the vessels whose outflow is impeded, raises arterial tension generally and causes an accentuation of the second aortic sound.

The changes in the aorta may so weaken its wall as to allow the formation of an aneurysm. For the same reason miliary aneurysms may be formed in the cerebral arteries and may result in rupture and cerebral hemorrhage.

Dry gangrene of the extremities may result from diminished supply of blood from the narrowing of the arterial lumen by connective tissue or thrombi.

Associated Lesions.—Contracted kidney occurs in the great majority of cases. It is often difficult to decide in a given case whether the arterial or the renal disease has been primary.

Emphysema with chronic bronchitis is present in a large number of cases, and the patient is liable to have cirrhosis of the liver, as alcoholism is a common factor in cirrhosis and in arterial sclerosis. Atheroma of the aortic valves is a frequent complication.

The **symptoms** are exceedingly diverse, depending upon which arteries are most affected and upon the secondary and associated lesions. Arterial sclerosis with compensatory hypertrophy is not inconsistent with general good health. When compensation fails the general symptoms of dilatation and heart-inefficiency occur.

Sclerosis of the coronary arteries may produce thrombosis with sudden death, fibroid degeneration, aneurysm or rupture of the heart, and angina pectoris.

Cerebral symptoms are those of cerebral endarteritis (which see), comprising acute and chronic degeneration, spasm of cerebral vessels with transient or permanent paralyses, and cerebral hemorrhages.

Renal symptoms may be absent, or the urine may be increased in quantity and of low specific gravity, with but occasionally hyaline casts and a trace of albumin. In other cases the renal symptoms are distinctly uræmic and may terminate the life of the patient.

The course of the disease may be complicated by aneurysm, by gangrene, or by an associated emphysema.

Physical Signs.—The combination of increased arterial tension, hypertrophy of the left ventricle, accentuation of the second aortic sound, and an appreciable thickening of the arteries affords conclusive proof of the existence of arterio-sclerosis. A high-tension pulse may exist with very little sclerosis, but sclerosis and high tension usually go together except when the left ventricle fails. The pulse-wave is slow in its ascent, is felt for an appreciably long period, subsides slowly, and between the beats the pulse remains firm and full. The wave-fluctuations are comparatively small. It is difficult, even impossible in some cases, to obliterate the pulse by firm pressure on the artery. The sphygmographic tracing (Fig. 19) shows a short slanting upstroke, a flat or rounded summit, and a gradual descent in which the dicrotic wave is slighly marked or absent.

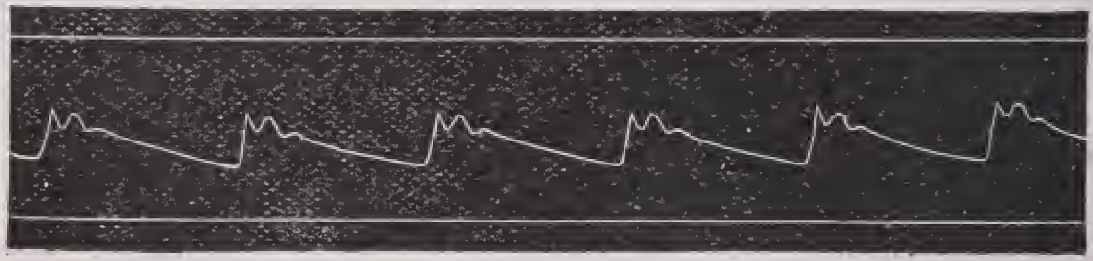
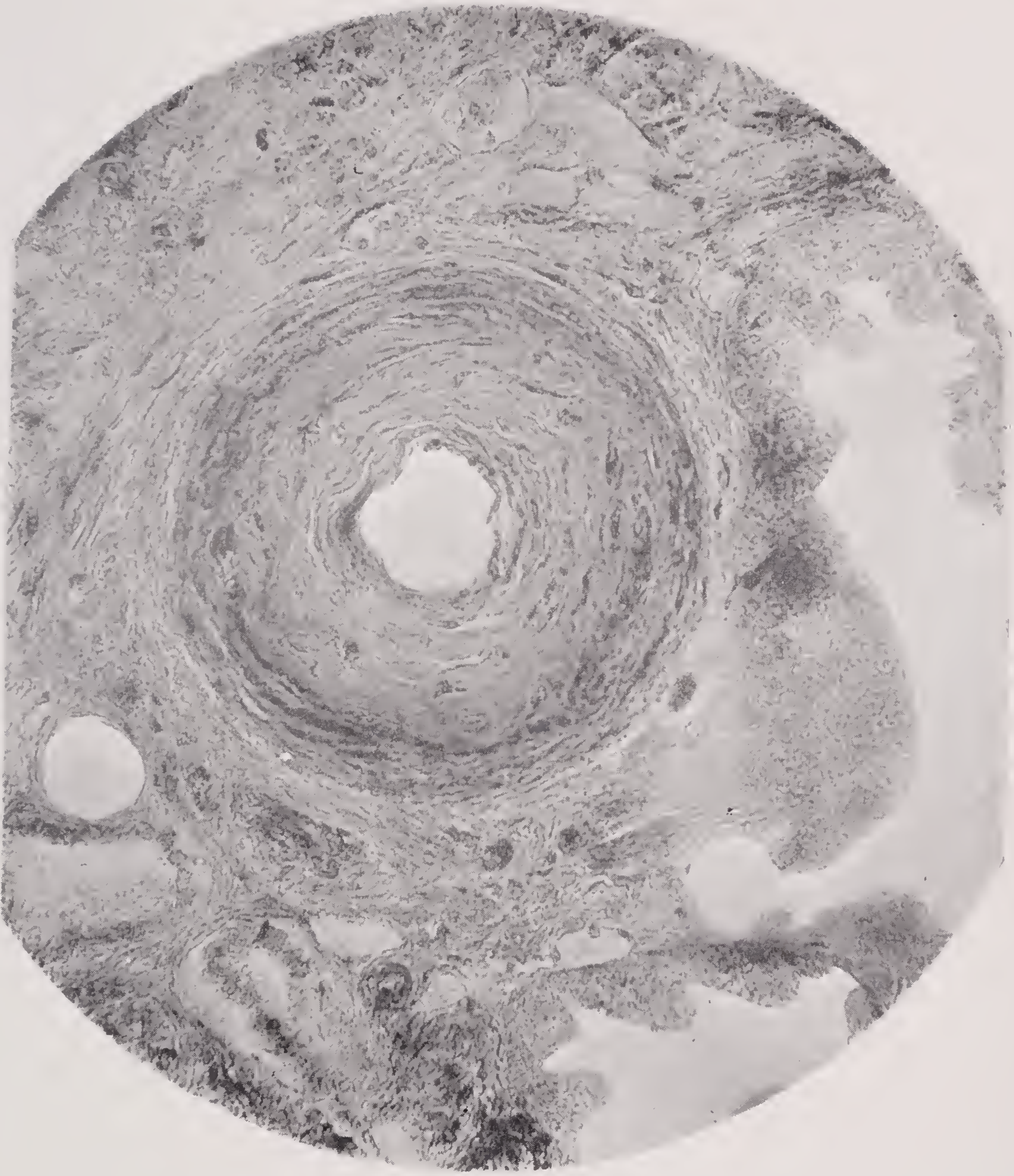


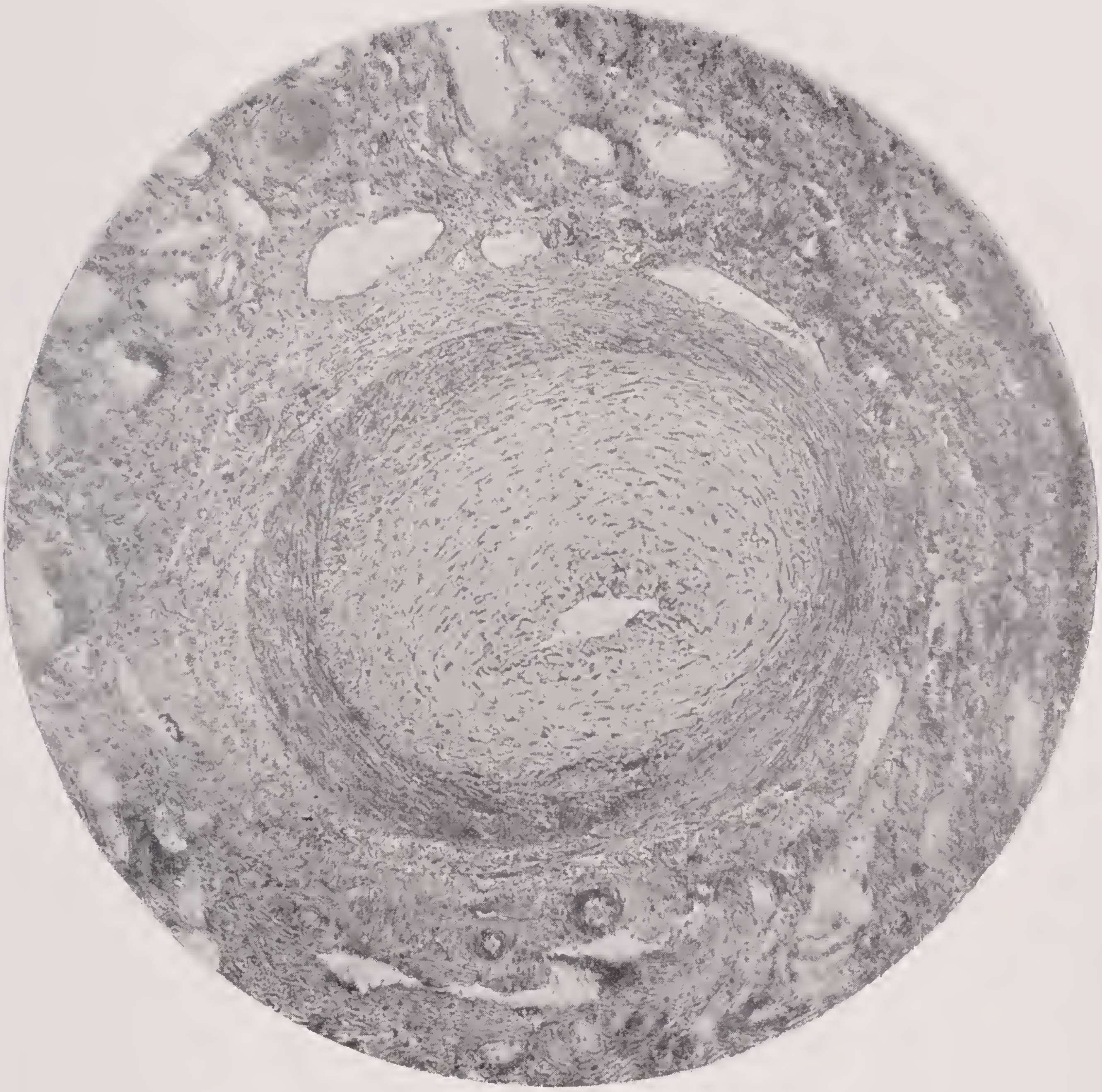
FIG. 19.—Sphygmogram showing high-tension pulse.

The **prognosis**, so far as life is concerned, is not unfavorable. The danger of renal or cardiac disease or of cerebral complications is always present. The chief question is whether compensatory hypertrophy of the left ventricle can be maintained. Sudden death may occur.

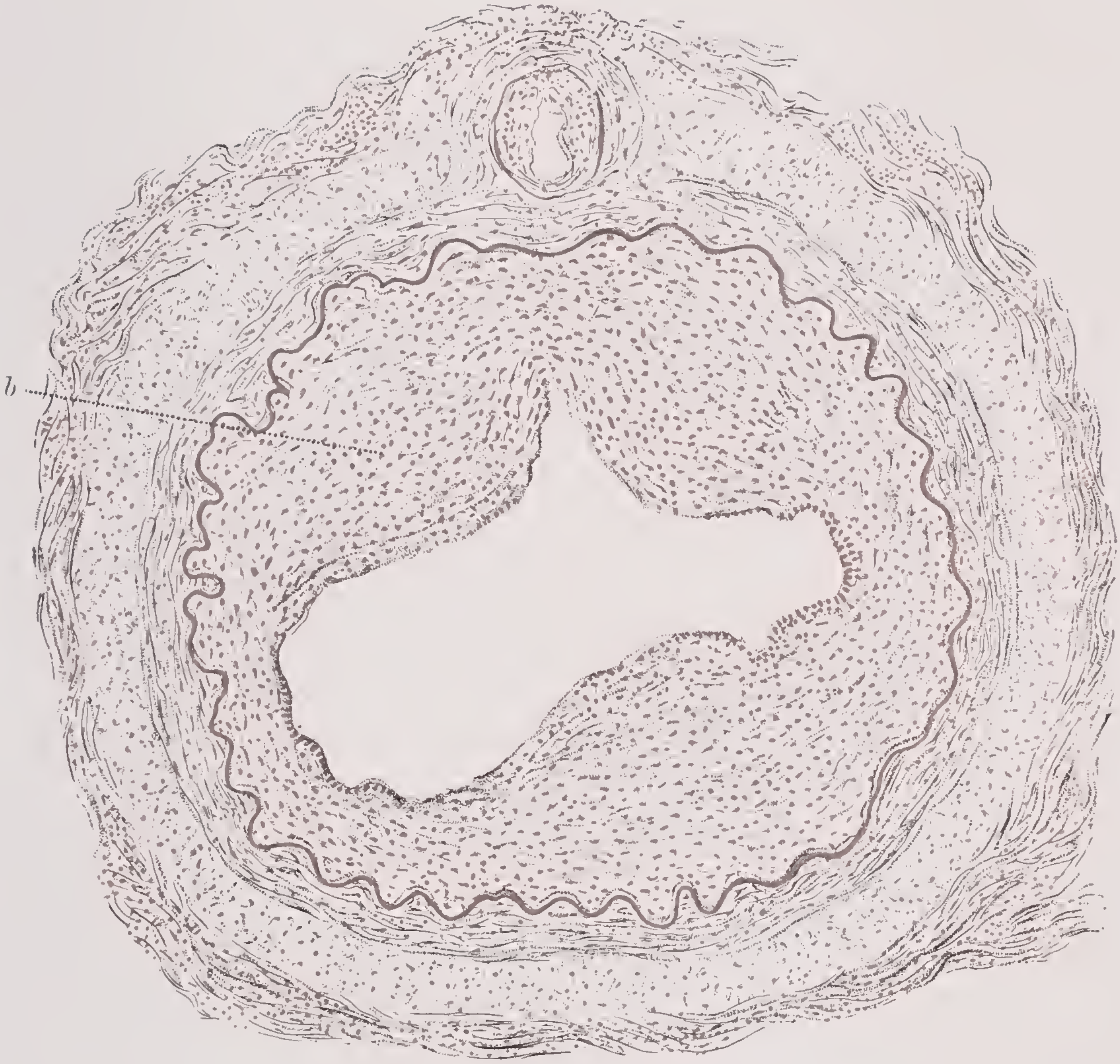
Treatment.—Much can be done to limit the extension of the disease by a quiet mode of life, plain, non-stimulating diet, and a correction of those conditions known to produce the disease. The state of the bowels and of the urine



Small artery: thickening of all the coats (Delafield).



Small artery: obliterating endarteritis (Delafield).



Syphilitic endarteritis (Finger) : *b* shows thickened intima.

should be regarded, and the skin should be kept active by daily baths. Alcohol is to be prohibited. High blood-tension should be reduced by appropriate drugs. Of these, nitroglycerin is the most serviceable, given in doses of gr. $\frac{1}{100}$ every three or four hours, or at longer intervals if not well borne. Iodide of potassium in gr. x doses t. i. d. is of service, especially in syphilitic patients. It is advantageously combined with choral in gr. v–x doses. Sudden and severe muscular efforts should be avoided, especially if the aorta be extensively involved. When the heart begins to dilate, stimulants will be required. Digitalis should not be given, however, unless its effect in raising arterial tension be balanced by its combination with nitroglycerin. Should acute dilatation occur, with lividity and dyspnœa, venesection may be resorted to.

SYPHILITIC ARTERITIS.

Besides syphilis being a causative factor in arterio-sclerosis and aneurysm, two specific forms of arteritis are described:

1. *Obliterating endarteritis* with proliferation of new tissue within the intima, obstructing the lumen. There is also a small-celled infiltration of the middle and external coats. This form of infiltration is not absolutely characteristic of syphilis, but should be so regarded should other syphilitic changes be found.

2. *Gummatous Peri-arteritis.*—Gumma develop within the adventitia, forming ovoid swellings along the course of the artery. There is usually an associated obliterating endarteritis. This process is distinctive of syphilis, and occurs especially in the coronary and cerebral arteries.

ANEURYSM.

The following forms of aneurysm occur:

1. *True* aneurysm, in which the sac is formed by the

arterial coats. The aneurysm may be cylindrical, fusiform, or sacculated.

2. *False* or *dissecting* aneurysm, in which, from laceration of the intima, blood makes its way between the layers and may rupture through the outer coats.

3. *Arterio-venous* aneurysm, where a communication exists between an artery and a vein. If there be an intervening sac, the term *varicose aneurysm* is applied; if the communication be direct, the condition is termed *aneurysmal varix.*

Etiology.—There is always some weakness of the arterial wall, so that it becomes dilated from the blood-pressure. There is almost regularly arterial sclerosis, and the conditions which produce this sclerosis are therefore causative factors of aneurysm. Bacterial infection of the aortic wall, producing aneurysm, has been observed with malignant endocarditis.

Embolism may lead to aneurysm by causing local degeneration or injury of the vessel-wall.

The determining cause of aneurysm is high arterial pressure from the arterial sclerosis or from severe muscular efforts. Aneurysm is more common in men than in women, and is more frequent among the working classes, as longshoremen, in whom alcoholism, syphilis, and over-work are important factors. It is rare before thirty and after fifty years of age, because arterial sclerosis does not appear before the earlier limit, and because muscular strain is not so common after the latter age.

Pathology.—The cavity of the aneurysm usually contains clots of blood, frequently laminated and partly organized. There may be calcareous degeneration of the clot. The vessels leading from the artery at the site of the aneurysm may be occluded; portions of the clot may become detached and carried into the circulation as emboli. Organization of the clot is conservative in its nature and is more common in sacculated aneurysm.

The aneurysm-wall is never composed of normal vessel-wall. The intima shows marked changes of arterial sclerosis. The media is changed, and is often in a condition of fatty degeneration. The adventitia is thickened by inflammatory processes, thus reinforcing the weakened arterial wall. The intima and the media may atrophy so that the wall consists of the adventitia alone.

Aneurysms vary in size from microscopic miliary aneurysms to those the size of a child's head or larger.

Aneurysms may rupture, may compress neighboring organs, and may cause pressure-erosion.

The situation of aneurysms varies. In 860 cases analyzed by Sibson, the situation of the aneurysm was as follows: The ascending portion of the arch, 141; the transverse portion of the arch, 120; the ascending and transverse portions together, 112; the sinuses of Valsalva, 87; the descending portions of the arch, 72; the transverse and descending portions together, 20; the whole arch, 28; the thoracic aorta, 71; the abdominal aorta at the cœliac axis, 131; the lower part of the abdominal aorta, 26.

Symptoms.—An aneurysm, being a pulsating tumor in the course of the arterial circulation, growing in the stiff-walled thorax, and having a tendency to rupture, naturally produces four groups of symptoms: (1) The presence of a growing pulsating tumor; (2) its pressure on surrounding parts; (3) its effect upon the circulation of the blood; (4) the symptoms due to erosion and rupture.

SYMPTOMS OF ANEURYSM OF THE THORACIC AORTA.—Pressure-symptoms are usually marked, and afford data for the localization of the aneurysm.

1. *Pressure on the Vena Cava or its Branches.*—There may be congestion and œdema of the arm and the face on

one side, more rarely on both; or in old-standing cases there may be a brawny swelling of the base of the neck, termed by the French "the collar of flesh." Large aneurysms of the ascending aorta sacculated downward may press on the inferior vena cava, causing œdema of the feet and ascites. There may be erosion from pressure and rupture into the superior vena cava with aneurysms of the ascending arch. Congestion of the chest-wall occurs from pressure on the azygos vein.

2. *Pressure on the Trachea and the Bronchi.*—Moderate pressure causes symptoms of inflammation and cough with expectoration which may contain blood. More marked pressure on the trachea causes inspiratory dyspnœa, either steady or paroxysmal, or orthopnœa. There are developed symptoms of gradual asphyxia. In some cases sudden fatal asphyxia may occur either from pressure-erosion of a tracheal ring, allowing the trachea to collapse suddenly like a membranous tube, or from the lodgement of a plug of mucus at the pressure-point, causing total obstruction. Tracheal compression is usually seen with aneurysms of the transverse arch. Compression of a bronchus causes localized bronchial catarrh with sibilant and sonorous râles, dyspnœa, and diminished breathing over that part of the lung. There may be bronchiectasis and suppuration of the lung. The left bronchus is the one more frequently compressed. With large aneurysms the lung may be partially compressed.

Rupture may occur into the lung, the trachea, or the bronchi. A large hemorrhage will cause death by anæmia, or, filling the bronchi, cause asphyxia or septic bronchopneumonia. Small repeated hemorrhages may occur from moderate leakage. Aneurysms of the descending arch often rupture into a pleural sac.

3. *Pressure on the œsophagus* occurs with aneurysms of

the descending aorta, more rarely with those of the transverse arch. Dysphagia may be either steady or paroxysmal, and may lead to great emaciation. The case may resemble one of œsophageal stricture, and care should always be taken in such cases to exclude thoracic aneurysm before passing an œsophageal bougie, as otherwise instrumental rupture of the aneurysmal sac may result. Rupture into the œsophagus may occur and may be the first symptom of an unsuspected aneurysm.

4. *Pressure or Traction on the Recurrent Laryngeal Nerve.*—The left side is more commonly affected. There may result spasm or paralysis of one or of both vocal cords. There is dyspnœa which is steady or paroxysmal; a brassy or clanging cough quite distinctive; husky or whispering voice or aphonia. Spasmodic dyspnœa of laryngeal origin is differentiated from obstructive dyspnœa of tracheal pressure by being relieved by inhalations of chloroform.

5. *Pressure on the Sympathetic Nerve.*—In the early stages the pupil on the affected side is dilated and the skin is paler than normal; later occur contraction of the pupil and flushing and sweating of the skin.

6. *Pressure on the brachial plexus* causes neuralgic pains, twitchings, and later areas of anæsthesia.

7. *Pressure on the Bones.*—Pressure on the vertebræ causes the erosion of their bodies with a steady boring pain in the back that is distinctive of aneurysms of the descending aorta. Complete erosion will expose the spinal cord to pressure with the symptoms of a transverse myelitis.

8. *Pressure on the intercostal nerves* causes neuralgic pain. Pressure on the sternum and the ribs is common with aneurysms of the ascending arch. Boring pain is experienced, and there is apparent a large pulsating tumor covered finally only by red shiny skin resembling that of a pointing abscess. External rupture is the inevitable result.

9. Aneurysms at the root of the aorta frequently cause angina pains.

Physical Signs.—*Inspection* in many instances is negative. There may be bulging of the chest-wall, best appreciated with oblique light. This bulging occurs usually above the third rib, to the right of the sternum. Aneurysms of the ascending aorta are found projecting in the left scapular region. An external tumor may be formed by the sac approaching the surface, invading the intermediate structures. The heart's apex is often displaced downward and to the left.

Palpation reveals an expansile pulsation of the tumor. There may be a heaving impulse without the appearance of an external tumor. There may be a systolic thrill, and in some cases a diastolic shock which is highly distinctive. Fluctuation may be detected when the sac has perforated the chest-wall. Care should be taken, however, in manipulation to avoid rupture of the sac. There may be pulsation in the sternal notch in case of aneurysm of the transverse arch.

Percussion reveals dulness or flatness whenever the aneurysm is large enough to approach the chest-wall. Small, deeply-seated aneurysms do not, therefore, yield dulness. Dulness on the right side of the manubrium indicates aneurysm of the ascending arch; dulness in the middle line extending to the left points to aneurysm of the transverse arch; while dulness to the left of the spinal column occurs with aneurysm of the descending aorta. There may be tenderness on percussion, and a sense of abnormal resistance.

Auscultation may yield negative results even with large aneurysms. There may be a continuous hum louder at each systole, or a systolic or double murmur. The aortic second sound is usually accentuated, or it may be replaced by the murmur of an associated regurgitation at the aortic valve. There may be a systolic murmur heard over the trachea, due to the expulsion of air at each pulsation of an overlying aneurysm.

The *pulse* in the arteries beyond the aneurysm is frequently altered, becoming slowed and the wave being partially effaced.

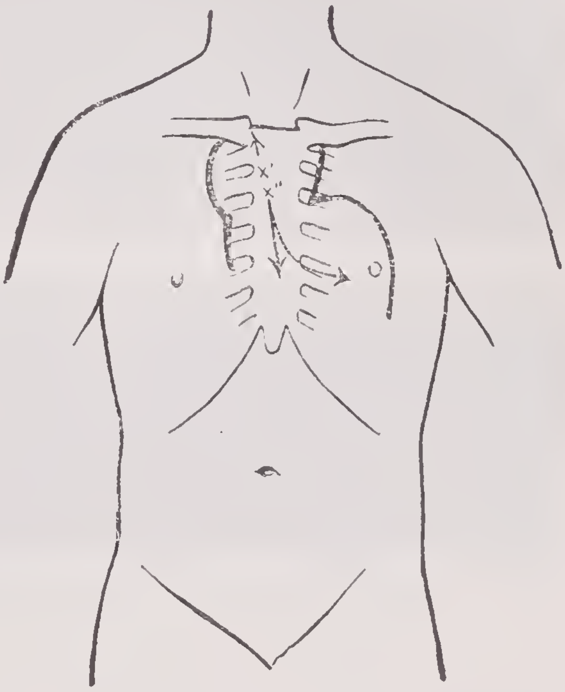


Fig. 20.—Aneurysm of the ascending aorta, showing shape of the outline and the position of the customary double murmurs.

Aneurysms of the ascending arch alone, delay all pulses equally. Large aneurysms of the descending aorta may totally efface the pulse-wave in the abdominal aorta and femorals. When the aorta at the origin of the innominate artery is involved, the right radial pulse is more retarded and effaced than the left. When the transverse arch is involved beyond the innominate, it is the left pulse that is the more affected.

Tracheal tugging is a sign of much value in detecting deep aneurysms pressing backward upon the trachea or the left bronchus. The patient should sit with the chin depressed, so as to relax the tissues of the neck. The examiner, standing behind the patient, raises the cricoid cartilage on the tips of the index fingers. If an aneurysm be present in the situation above noted, a characteristic downward tugging will occur with each pulsation. This tugging is a sign of great value, although not absolutely pathognomonic. The heart may be displaced downward and to the left. It is not usually enlarged unless from some coexisting lesion.

Diagnosis.—Throbbing of the aorta in aortic insufficiency, and displacement of the aorta forward with spinal curvature, may simulate aneurysm, but pressure-symptoms, pain, and retardation of the pulse are absent. Sacculated empyema receiving impulses from the heart may cause a pulsating tumor, but the pulsation is not expansile, there are no circulatory symptoms, and septic symptoms occur.

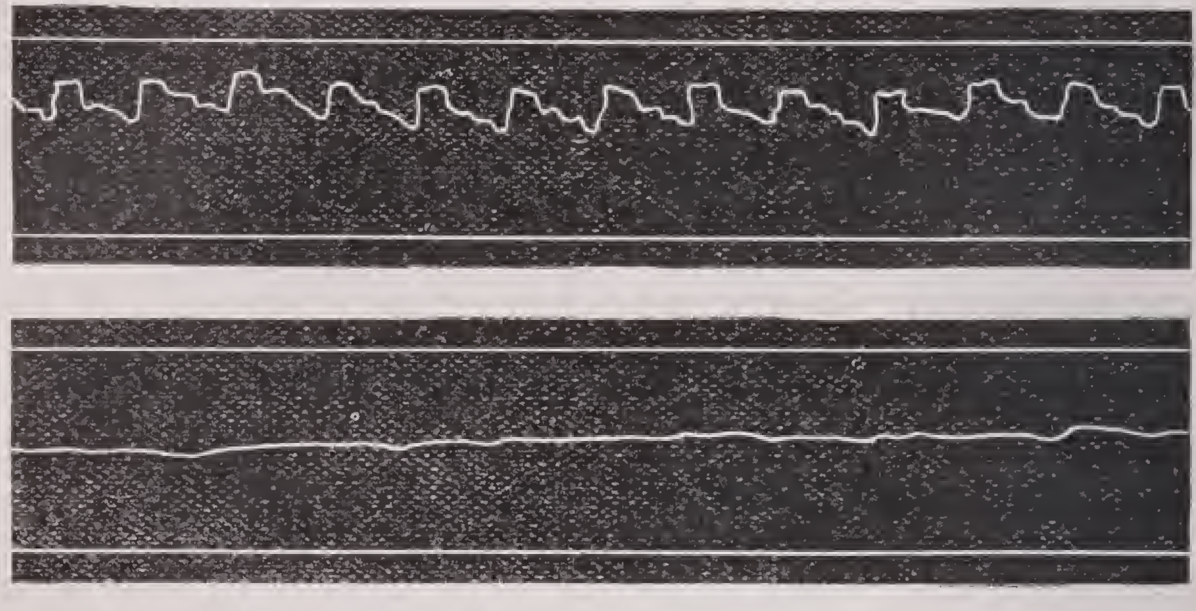


FIG 21.—Sphygmograms of the radial pulse on the right (*a*) and the left side (*b*), from a case of aneurysm of the transverse part of the arch of the aorta.

Pulsating sarcoma and other growths of the mediastinum often present great difficulties in diagnosis. The pulsations in these growths are not expansile, as in aneurysm, have less force and power, and have no diastolic shock. In some cases a differential diagnosis between a pulsating tumor and an aneurysm is impossible.

The **prognosis** is always grave. Recovery may occur, but it is not to be expected. The aneurysm may rupture at any time, and the rupture may even be the first symptom. Dissecting aneurysms just above the sinuses of Valsalva rupture usually into the pericardium, causing sudden death. Sudden heart failure is common with aneurysm without rupture, and death may result from obstructive dyspnœa, myelitis, dysphagia, and exhaustion, or from associated endocarditis of the aortic valves. The course of the disease is usually about two years, although life may be prolonged in some cases for five or ten years.

The **treatment** of aneurysm of the thoracic aorta consists in the attempt to secure coagulation within the sac. Rest is an essential feature of the treatment, and should be as nearly absolute as possible. Mental excitement of all kinds should be avoided. Tufnell's treatment consists in the enforcement of rest and a restricted diet. He allows for breakfast 2 ounces of bread and butter with 2 ounces of milk; for dinner, 2 or 3 ounces of bread and 2 or 3 ounces of meat, with from 2 to 4 ounces of milk or claret; for supper, 2 ounces of bread and 2 ounces of milk. This plan succeeds best in small sacculated aneurysms. Few patients, however, can stand such a radical reduction of food, so this treatment cannot be enforced rigorously. A more liberal supply may be given, but fluids should be restricted as much as possible. Systematic bleeding may prove of service in the earlier stages in robust subjects: 8 or 10 ounces of blood may be abstracted every ten days or two weeks, provided that excessive anæmia is not produced.

Of medicines, iodide of potassium is most commonly employed, the aim being to tranquillize the circulation and reduce blood-tension without increasing the frequency of the pulse. To find the proper dose the patient should be put to bed for several days, to find the rapidity of the pulse at rest. Then the iodide is to be given in 5-grain doses, well diluted, three times a day, and gradually increased as long as the pulse is not made more frequent. Rarely more than 10 or 15 grains three times a day are necessary. One marked effect of the iodide is the reduction of pain.

Aconite may be used for temporary over-action of the heart, but its administration for any length of time is not recommended.

Various forms of local treatment have been recommended, but with indifferent success. The insertion of horse-hair, catgut, wire, and the injection of styptics into the sac have been tried. Loreta's method has been followed in some cases by good results. This treatment consists in filling the sac with fine silver wire pushed through a hypodermic needle, combined with electrolysis.

Special symptoms are to be treated as they arise. Dyspnœa and congestion may be relieved by timely venesection. Morphine is to be employed for pain. Urgent dyspnœa may seem to indicate a tracheotomy, but the operation is usually useless, as the obstruction is below the site of the operation in nearly all cases. If chloroform inhalations relieve the dyspnœa, and if laryngoscopic examination reveals bilateral abductor paralysis, the operation may be resorted to. External rupture is to be retarded by painting the surface with a solution of gutta-percha, by the use of ice-bags, or by a metal or an elastic support.

ANEURYSM OF THE ABDOMINAL AORTA.

The usual situation of aneurysms of the abdominal aorta is near the cœliac axis, which is frequently involved. The aneurysm may be fusiform or sacculated, and may be multiple. It may project backward, eroding the vertebræ, or it may project forward, attaining considerable size. It may rupture into the pleura, the retroperitoneal tissues, the peritoneum, or the intestines.

Symptoms.—Pain is the most prominent symptom; it may be sharp, shooting, radiating down the legs, or it may be the steady boring pain of bone-erosion. Gastric symptoms are common, especially gastralgia and vomiting. Embolism of the mesenteric artery may occur, producing severe colicky pain. Paræsthesia and paraplegia may result from pressure on the cord.

Physical Signs.—There may be a pulsating tumor apparent on inspection. By palpation a tumor of expansile pulsation is appreciated. There may be heard a systolic, diastolic, or double murmur. The pulse in the femorals is retarded and may be obliterated. To avoid mistaking a throbbing aorta for aneurysm, Osler says, "It is to be remembered that no pulsation, however forcible, nor the presence of a thrill or a systolic murmur, justifies the diagnosis of abdominal aneurysm unless there is *a definite tumor which can be grasped and which has an expansile pulsation*." A tumor of the pylorus with an impulse transmitted from the underlying aorta may be mistaken for aneurysm, but the impulse is not expansile and is lost when the patient assumes the knee-chest position, the tumor falling forward away from the aorta.

The **prognosis** of abdominal aneurysm is grave, although recovery is not impossible.

The **treatment** generally is that of thoracic aneurysm. Pressure on the proximal portion of the aorta may be resorted to under chloroform. Pressure should not be too severe, as bad results have followed the bruising of the sac.

III. DISEASES OF THE RESPIRATORY SYSTEM.

1. DISEASES OF THE LARYNX.

SPASM OF THE LARYNX.

Two forms of spasm of the larynx are recognized: (1) Laryngismus stridulus; (2) Spasmodic laryngitis.

1. Laryngismus Stridulus (Thymic Asthma).—This form occurs in children under two and a half years of age. Rickets is found in two-thirds of the cases. It is more common in boys, in delicate, dyspeptic, and nervous children. It is less frequent in America than in England, France, or Germany. It may be a symptom of tetany.

Etiology.—The attack may be induced by any reflex cause for irritation, such as dyspepsia, poor air, constipation, dentition, or attacks of crying; it is favored by inflammation of any part of the respiratory tract.

Pathology.—The disease consists of a spasm of the adductors of the larynx, without inflammatory basis.

Symptoms.—The attacks may come at any time, but they are most common just as the child awakes. Respiration is suddenly arrested; the child struggles for breath; the face is pale, cyanotic, or congested; the pulse becomes weak and flickering; after a number of seconds the spasm relaxes and air is inspired with a loud crowing sound. During the attack there may be spasm of the hands and feet ("carpopedal spasms"), or even general convulsions. The attack is not accompanied by cough, hoarseness, or fever. The paroxysms may be as frequent in severe cases as thirty or forty during the twenty-four hours, and they may be continued at intervals for months. Slight reflex causes may at any time bring on the paroxysms.

The **prognosis** is generally good, but it is possible for the child to die in any of the attacks. Severe spasms may be the cause of meningeal hemorrhage.

Treatment.—The spasm is of such short duration that there is but little time for treatment. It seems best not to shake the child nor to dash water in its face, as has been recommended, but to keep the child quiet until the attack is over. Should the apnœa be persistent, a hot bath (95° F.) should be given and a cold compress be applied to the head. Intubation may be resorted to if danger be imminent.

Recurrences of the paroxysms may be prevented by rectal injections of chloral hydrate (gr. ij–v) in milk of asafetida (ʒij), to which from 2 to 5 grains of sodium bromide may be added. Swollen gums should be lanced freely. The bowels should be regulated, and attention be paid to the proper scientific feeding of the child. Osler recommends warm baths two or three times a day while the back and the chest are being sponged with cool water. Any cause for reflex irritation should be discovered and properly treated. The importance of good fresh air and sunlight must be remembered in all cases.

2. SPASMODIC LARYNGITIS (SPASMODIC CROUP).—This is the more common form of spasm; it occurs regularly in children between two and five years of age, and equally in strong and in delicate subjects.

Symptoms.—The attack comes regularly after the first heavy sleep, usually between 1 and 3 o'clock A. M. The child suddenly awakes and sits up in bed with evident dyspnœa. The inspirations are noisy, difficult, and "croupy;" the voice is husky; there is a brassy "croupy" cough. The oppression and cyanosis may appear alarming, but after a time (from half an hour to an hour) the attack suddenly ceases and the child is as well as ever. During the attack there are neither constitutional nor inflammatory symptoms, and there is no fever. The attack may be repeated on subsequent nights, but in these cases there is usually added a mild catarrhal laryngitis, with cough, hoarseness, and possibly slight fever, during the day.

The **prognosis** is perfectly good, notwithstanding the alarming appearance of the child.

Treatment.—A prompt emetic should at once be administered, such as wine of ipecac ʒj, or mustard and water; the yellow sulphate of mercury (gr. ij–v) has been recommended, but as it may cause gastro-enteritis it should not be used. If the attack persist, hot baths and hot fomentations to the throat may be employed.

ACUTE CATARRHAL LARYNGITIS.

Etiology.—Many cases of acute catarrhal laryngitis are due to catching cold or to breathing irritating and impure air, especially in patients with catarrhal affections of the nose and the throat. An attack may be induced by over-use of the voice. Laryngitis may be one of the lesions of "the grippe" or of measles; it may also complicate any disease of the bronchi and lungs attended with cough and expectoration. It may also occur with any of the acute infectious diseases.

Pathology.—On laryngoscopic examination the mucous membrane of the larynx is seen to be red, congested, and swollen. The inflammation may be generally distributed or it may involve only certain areas. The vocal cords are usually involved, and their mobility is impaired. There may be an over-secretion of the mucous glands, or only a slight mucoid exudation. Superficial ulcerations may result in severe cases. In certain cases in adults there may be considerable œdema of the larynx, which may constitute a formidable complication.

Symptoms.—*In adults* the course of the disease is somewhat different from that in children. Fever may be slight or absent, but malaise to some extent is common; slight rigors may initiate a severe attack. The voice becomes hoarse or is reduced to a whisper; the cough is croupy or barking, and is usually paroxysmal and harassing. Pain referred to the larynx may be quite severe and steady; it is usually increased by swallowing, talking, or coughing. In other cases a burning, irritating feeling alone is complained of. Pressure over the cricoid cartilage may cause pain or a paroxysm of coughing. In severer cases with œdema of the glottis dyspnœa may be a marked symptom. The difficulty of breathing may be continuous or paroxysmal; it may lead to asphyxia. This complication is more common in those suffering from alcoholism and Bright's disease.

In Children.—The peculiarity of laryngitis in children is that it is often complicated by spasm of the larynx, constituting a clinical group of symptoms to which the names "pseudo-croup" and "catarrhal croup" have been given. This complication usually occurs in the winter months and in children under three years of age, although some children continue to have attacks until their tenth or twelfth year. The symptoms resemble those in adults, but they are more marked at night. The child is usually awakened by severe and distressing dyspnœa, not, however, of the same explosive violence that is seen in pure spasmodic laryngitis. The attack wears itself out in an hour or so, and the child is left with only the ordinary symptoms of laryngitis.

The **diagnosis** must be made from spasmodic laryngitis and membranous croup. In spasmodic laryngitis the attack begins suddenly with explosive violence, and between the attacks the child is well and has no fever. In catarrhal laryngitis the child has been ailing, the dyspnœa begins more gradually, and between the attacks of dyspnœa the child has a cough, the voice is hoarse, and there is some fever.

In membranous laryngitis the dyspnœa is more continuous, the general symptoms are more severe, and there are usually patches of membrane on the pharynx and the tonsils. The cervical glands are also swollen.

In adults laryngoscopic examination will make a positive diagnosis from nervous or hysterical aphonia and from simple œdema glottidis.

Treatment.—The patient should breathe warm, pure, and moist air, and must avoid over-use of the voice. Cold applications to the neck are often of great service. The following prescriptions are of use in allaying the cough:

℞. Ammonii chloridi, ʒiss;
Mist. glycyrrhizæ comp., ℥iv.—M.
Sig. A teaspoonful every two hours in a wineglassful of water for an adult.

℞. Antimonii et potassii tartratis, gr. $\frac{1}{50}$;
Codeiæ, gr. $\frac{1}{8}$.—M.
Ft. chart. No. j.
Sig. One every two or three hours for an adult.

The inhalation of menthol and compound tincture of benzoin is of great service. When the acute stage is passed astringent sprays may be employed.

Attacks of dyspnœa in children are to be treated as are those of spasmodic laryngitis.

CHRONIC CATARRHAL LARYNGITIS.

Etiology.—This condition may result from repeated acute attacks, from breathing irritating dust or vapor, from excessive smoking, and from over-use of the voice; it may complicate chronic bronchitis and pulmonary phthisis. The most common cause, however, is chronic nasal and pharyngeal catarrh, especially if the nares be occluded and unfit for proper nose-breathing.

Pathology.—The mucous membrane of the larynx is congested and thickened, and the proper motility of the vocal cords is impaired. Secretion is excessive or scanty and tenacious.

Symptoms.—There is a tickling, irritating cough, worse at night and in damp weather. The voice is husky, has no "reaching power," and may at times be lost entirely. Tickling sensations in the larynx cause a constant desire to clear the throat.

Treatment.—The first thing is to remove the cause of the laryngitis, and especially to direct proper treatment to nasal and pharyngeal catarrhal conditions, should they exist. Heated rooms and impure air are to be avoided, smoking is to be prohibited, and proper exercise in the open air is to be insisted upon. Mufflers and neck-handkerchiefs should be avoided. Of great service are astringent sprays, especially nitrate of silver (gr. x : ℥j). Obstinate cases should be referred to a throat specialist.

MEMBRANOUS LARYNGITIS.

Etiology and Synonym.—The occurrence of a membranous laryngitis not diphtheritic in its nature is denied by some authors; but cases undoubtedly occur in which the Klebs-Loeffler bacillus cannot be demonstrated, but in which streptococci and staphylococci play a causative rôle. Further bacterial examinations, however, are desirable to place these cases of non-diphtheritic membranous laryngitis upon a firm and independent basis. *Synonym:* Membranous croup.

The **pathology** is practically that of diphtheria of the larynx, except that the deeper tissues are not, as a rule, so extensively infiltrated, and that the Klebs-Loeffler bacilli are not present. The exudation may be confined to the larynx, or it may spread to the trachea and bronchi, and more rarely to the pharynx, the palate, and the tonsils. In the exudate staphylococci and streptococci are found. The disease occurs in children between two and seven years of age almost exclusively.

The **symptoms** of the non-diphtheritic laryngitis closely resemble those of the diphtheritic form, but they lack the extreme prostration so characteristic of diphtheritic toxæmia. The points of differential diagnosis from diphtheria are as follows: (1) The patient is a child between two and seven years of age; (2) there is no history of exposure to diphtheria; (3) the patient does not act as a source of contagion to others; (4) albumin and casts are not apt to be found in the urine; (5) the symptoms are those of laryngeal obstruction and inflammation rather than those of obstruction, prostration, and sepsis; (6) the lesion occurs in a primary form in the larynx, whereas in diphtheria the membrane on the larynx is almost invariably secondary to membrane on the pharynx, the tonsils, or the palate; (7) cardiac failure, peripheral neuritis with paralysis, and nephritis are not observed as sequelæ.

Treatment.—Owing to the necessary uncertainty of diagnosis, the case should be isolated thoroughly from the start, and be considered as diphtheria. The treatment is that of laryngeal diphtheria in every particular, except that pharyngeal irrigation need not be insisted upon.

TUBERCULAR LARYNGITIS.

Etiology and Synonym.—Tubercular infection of the larynx is almost always secondary to pulmonary tuberculosis, in which it complicates from 18 to 30 per cent. of all cases. The larynx may be involved early or late in the course of the pulmonary disease. The occurrence of tubercular laryngitis is more common in men than in women, and between the twentieth and thirtieth years. *Synonym:* Laryngeal phthisis.

Pathology.—The mucosa is thickened by tubercular deposits and by œdema, especially over the arytenoid cartilages. Tubercles appear upon the surface, and often coalesce to form masses, which may ulcerate. The resulting ulcers are broad and shallow, having grayish bases and being surrounded by thickened mucosa. There may be a destruction of the deeper tissues by extension of the ulceration. The disease may spread to the pharynx, the epiglottis, or the œsophagus.

Symptoms.—Hoarseness or aphonia constitutes an early and a constant symptom, to which, however, no diagnostic importance can be attached, as the change of voice may be due to muscular insufficiency of the vocal cords or to chronic catarrhal laryngitis, to which conditions phthisical patients are extremely susceptible. There is usually an annoying painful cough, which is, however, sometimes absent even in aggravated cases. Pain on talking may be severe, and neuralgic pains running to the ears may be complained of. Pain on swallowing may be so distressing that

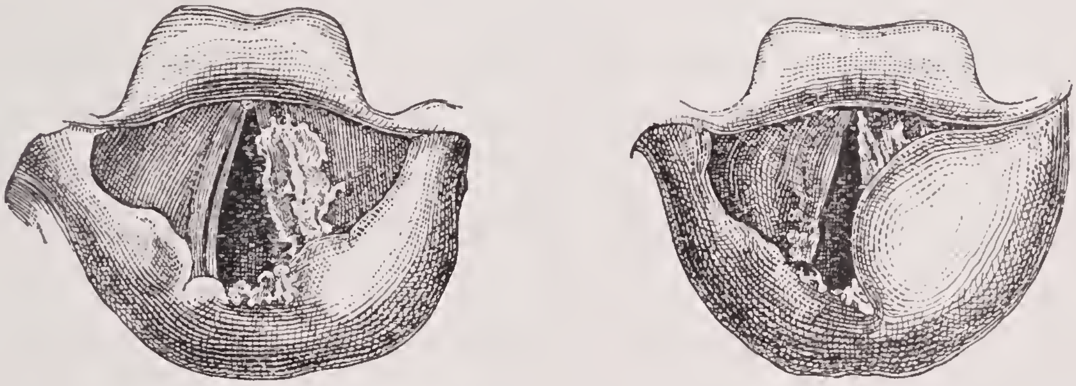


FIG. 22.—Tubercular laryngitis (Brown).

the patient is with difficulty prevailed upon to take sufficient food; this pain usually results from tubercular involvement of the epiglottis or the pharynx. Dyspnœa may appear late in the disease, in either a constant or a paroxysmal form, often necessitating tracheotomy to avert death from suffocation or to render the patient's condition a trifle more endurable.

The **diagnosis** is made by laryngoscopic examination. In the earlier stages of the disease the larynx is of a peculiar pallor, while the arytenoids show a characteristic club-like swelling. Later in the disease the tubercular masses and ulcerations are easily recognized. A diagnosis is aided by finding tubercular changes in the lungs, and is rendered certain by the presence of tubercle bacilli in the secretion from the base of the ulcer.

The **prognosis** for duration of life depends largely upon the primary pulmonary disease. Death may be hastened by suffocation, inanition, or exhaustion.

Treatment is often unavailing. The larynx should be kept free of secretion by cleansing sprays. Astringents may be used by spray or insufflation, and powdered iodoform may be insufflated with benefit. Pain in swallowing may be controlled by spraying the throat with a 2 per cent. solution of cocaine. Applications of lactic acid and submucous injections of creosote have been recommended warmly, but they should be used only by skilled specialists. For the relief of the dyspnœa tracheotomy may be indicated. Surgical treatment of tubercular laryngitis consists in the scraping out of tubercular deposits, the parts being exposed by a median thyroidectomy if necessary.

SYPHILITIC LARYNGITIS.

The *hereditary* form of syphilitic laryngitis usually appears in early childhood; it is characterized by the formation of gummata, deep ulcerations, and cicatricial deformities. The *acquired form* is more common.

As a *secondary* lesion the larynx may be the seat of an erythema, imparting to it a purplish-red, mottled appearance and giving rise to the symptoms of a simple laryngitis. Mucous patches and condylomata are rarely observed.

As a *tertiary* lesion the disease is most common. There is a chronic catarrhal laryngitis, with an infiltration of the mucosa with gummata varying in size from a pin's head to a hazelnut. These gummata may undergo resolution if the appropriate treatment be effectual, or they may ulcerate, frequently destroying the deeper structures. The lesions may involve the larynx or may be unilateral. Cicatrices follow the ulcerations, and they may be sufficiently extensive to cause great deformity. Laryngeal stenosis from such a cause is a frequent sequela.

Treatment must be prompt and energetic—mercury for the secondary form, potassium iodide in large doses for the tertiary period, under the rules laid down for the treatment of syphilis. The larynx must be kept clear by sprays, and any resulting stenosis is to be treated by cutting or by dilatation. Should the gummata be large enough to occlude the glottis, tracheotomy may be necessary to avert fatal dyspnœa.

ŒDEMATOUS LARYNGITIS.

Etiology and Synonym.—Œdema of the glottis may complicate severe acute inflammation, whether due to cold, to inhalation of irritant vapors, or secondary to certain of the acute infectious diseases. It may occur with perichondritis, as in tubercular or syphilitic laryngitis, or it may be due to the spread of intense inflammations of neighboring parts. Œdema of the glottis may suddenly occur in the course of acute or chronic Bright's disease, but this mode of occurrence is exceedingly rare. *Synonym:* Œdema of the glottis.

Symptoms.—Dyspnœa is suddenly developed and becomes rapidly urgent. The voice becomes husky and is finally lost. Respiration is accompanied by stridor. Symptoms of asphyxia occur as a terminal event.

The **diagnosis** can be made with certainty by laryngoscopic examination. The ary-epiglottidean folds are seen to be intensely œdematous, meeting in the median line in severe cases. The epiglottis may be involved by the œdema, and in rare cases the parts below the cords are also swollen. Without the aid of the laryngoscope the diagnosis can usually be made by feeling the œdematous parts with the finger-tip.

The **prognosis** is exceedingly bad.

Treatment.—No time should be lost in temporizing. Ice poultices should be applied to the neck, and after spraying the throat with cocaine long incisions should be made into the œdematous parts with a curved bistoury protected except at the extreme tip. Tracheotomy should be resorted to unless prompt relief is afforded by the scarifications.

2. DISEASES OF THE BRONCHI.

ACUTE CATARRHAL BRONCHITIS.

Etiology.—Primary bronchitis is exceedingly common as the result of "catching cold," beginning as a coryza and extending downward in the chest. It is more frequent in children and old people than in adults, and in the aged is a formidable disease. Cases are more common in the winter and spring months and in climates in which extreme and sudden changes in the weather occur. The disease is occasionally seen in localized epidemics, and it may even assume an apparently contagious nature. Those who lead an indoor life with insufficient ventilation are more susceptible, and those with "delicate chests" develop the disease upon the least provocation. It may be caused by the inhalation of noxious gases, such as ammonia, chlorine, or sulphurous acid, or by the breathing of smoke or of dust, especially in factories.

Secondary bronchitis occurs with measles, influenza, whooping-cough, and any of the infectious diseases, and with diseases of the heart, lungs, or pleura. The consideration of these cases is referred to their respective headings.

Pathology.—The lesion is bilateral, affecting the trachea and the larger and medium-sized bronchi. In severe cases, especially in children and old people, the smaller bronchi may be involved. The mucous membrane is congested, reddened, swollen, and covered with mucus and muco-pus containing desquamated epithelial cells and leucocytes. The mucous glands are enlarged. In severe cases the submucosa is œdematous and is infiltrated with leucocytes.

Symptoms.—*In Adults.*—There may be chilly feelings at the onset, followed by moderate fever, which rarely rises to 103° F. even in the severest cases. Heaviness and malaise are present, with general pains in the bones, and the patient may be sick enough to be in bed.

The chest symptoms set in with a feeling of tightness and oppression, a scraped feeling under the sternum, and a cough. At first the cough is dry, hoarse, and painful, coming in distressing paroxysms. Pain in the chest during coughing is chiefly felt along the attachments of the diaphragm and down the sternum. When secretion is established the cough is much relieved. The sputum is at first scanty and mucous in character; it may be blood-streaked; later it becomes more abundant and muco-purulent. Dyspnœa is not a regular feature of bronchitis of adults, although in some patients there may be asthmatic breathing.

Bronchitis *in young children* is a more serious disease, from its tendency to involve the smaller tubes and to merge into broncho-pneumonia. In young babies cough, fever, and rapid breathing may be the only symptoms. The rapid breathing makes it difficult to nurse these young infants. In older children the disease may be either mild or severe. In severe cases there may be convulsions at the outset. The fever is high (102° to 103° F.) with morning remissions; the pulse is rapid; the breathing is rapid and may be insufficient, as shown by duskiness of the skin and symptoms of carbon-dioxide poisoning. There is cough, but the sputa are regularly swallowed. There are apt to be gaseous distention of the stomach and vomiting. It is often hard to tell where bronchitis ends and broncho-pneumonia begins. If the symptoms be severe, if they continue for three or four days without improvement, and if the physical signs point to the involvement of the smallest bronchi, it is best to regard the case as one of pneumonia. To this class of cases the term "capillary bronchitis" is given, but they are more properly cases of broncho-pneumonia.

Bronchitis *in old people* is dangerous from the prostration and from the tendency of the disease to spread to the smaller tubes, and even to pass into broncho-pneumonia. There is a slight but irregular fever; prostration is extreme, the weakness interfering with the cough and the proper clearing out of the accumulated mucus. There is no complaint of dyspnœa, but the breathing is rapid and often is insufficient. There may be slight delirium, especially at night.

Physical Signs.—The regular physical signs of bronchitis consist of coarse large and small mucous râles. In children bronchial fremitus is usually present. In the earlier stages there may be sibilant and sonorous breathing. Subcrepitant râles point to the involvement of the smaller tubes.

Râles may be absent (1) in mild cases with scanty secretion, (2) in inflammation of the trachea and the large bronchi alone, (3) after coughing with the expulsion of the secretion, and (4) in the feeble and aged, in whom respiration is too feeble to generate râles.

The **prognosis** is good except in young infants and in old people, in whom broncho-pneumonia may develop. In the aged death from exhaustion may occur.

The **diagnosis** of bronchitis is simple, but it is not to be considered complete unless it be determined whether the bronchitis is primary or is dependent upon some other disease, or whether it be an acute exacerbation of a chronic bronchitis. Bronchitis at an apex implies tubercular disease. Bronchitis of the finer tubes, worse at the apex and with continual high temperature, suggests miliary tuberculosis. Unilateral bronchitis suggests aneurysmal pressure, chronic interstitial pneumonia, or pleural adhesions. Bronchitis with dyspnœa and repeated slight hemorrhages suggests tuberculosis or cardiac disease. Recurring attacks of bronchitis in young children suggest tuberculosis or tubercular bronchial glands.

Treatment.—In the majority of cases no treatment is required. Much can be done to abort an attack by free diaphoresis at the onset. A Dover's powder at night with a hot mustard foot-bath often suffices. A hot lemonade containing a little whiskey is a popular and efficacious remedy. Turkish baths are objectionable from the danger of catching more cold on leaving the bath-house, but a hot bath at night is often useful if the patient need not leave the house afterward. The bowels should be opened by salines, and 10 grains of quinine in a single dose may be given. Delicate patients should be kept indoors or in bed. Expectorants are very useful to promote free secretion, but they are often given indiscriminately. They may be combined with sedatives. In the dry stage tartar emetic in gr. $\frac{1}{50}$ doses every two hours until slight nausea is experienced is often serviceable, but preparations of squills, senega, ipecac, or ammonium muriate may be used. The sedatives employed are codeia and dilute hydrocyanic acid or small doses of opium. The following formula is recommended :

℞. Codeiæ,	gr. $\frac{1}{6}$;
Ammon. muriat.,	gr. ij ;
Acid. hydrocyanic. dilut.,	♏ iij ;
Syrup. scillæ comp.,	♏ x ;
Infus. prun. Virg.,	ad ʒj.—M.

Sig. Dose every two or three hours for an adult.

When free secretion is established, ammonium muriate with mist. glycyrrhizæ comp. is of much benefit. The paroxysmal cough of the earlier stages is frequently benefited by the inhalation of steam, to which may be added compound tincture of benzoin or terebene. If the disease threaten to become chronic, the patient should be put upon a supporting tonic treatment, and, if possible, be sent away to some warm, dry, inland place.

Bronchitis in children is to be treated like that in adults, except that opiates are not well borne. Severe cases are to be treated like broncho-pneumonia. If the bronchi become blocked by a too profuse secretion, interfering with free respiration, a simple emetic should be given. Bronchitis in old people frequently requires stimulation by alcohol, digitalis, and strychnine. Opiates are to be used with extreme caution.

ACUTE CROUPOUS BRONCHITIS.

Etiology and Synonyms.—The disease is rare. It may occur as the result of breathing irritating vapors and steam, but it is more often secondary to diphtheritic or croupous laryngitis by extension downward. *Synonyms:* Acute fibrinous bronchitis; Acute membranous bronchitis; Diphtheritic bronchitis.

The **lesion** is that of a croupous inflammation of a mucous membrane. In cases complicating laryngeal diphtheria the Klebs-Loeffler bacilli are present in the membrane.

Symptoms.—There may be a chill with fever and prostration at the onset, or there may be the previous history of membranous laryngitis. When developed, there is a cough with muco-purulent expectoration. From time to time there will be attacks of paroxysmal cough with the expectoration of rolled-up casts of a bronchus and its branches. These casts consist chiefly of coagulated fibrin which in places has undergone hyaline degeneration, and in its meshes are leucocytes, epithelial cells, and frequently Leyden's crystals and Curschmann's spirals. These casts readily unroll in the water. During the coughing attack suffocation seems imminent, and often some blood is raised, but after the cast is expectorated the distress abates. Between the coughing attacks the symptoms resemble severe acute bronchitis, frequently with symptoms of insufficient aëration of the blood and of dyspnœa.

The **physical signs** consist of bronchial râles of all kinds, fine and coarse, with sibilant and sonorous breathing. When a bronchus is occluded, breathing sounds may be absent over that portion of lung, returning, however, when the occluding membrane is coughed up.

The **prognosis** is bad, depending on the nature of the primary disease and the extent of the bronchi involved. Death usually occurs from suffocation. It is a most serious complication after tracheotomy.

The **treatment** is that of acute bronchitis, except that inhalation of steam is often of service in loosening the false membrane. For this purpose hypodermics of pilocarpine may also be employed. Inhalations of pure oxygen gas are of benefit if symptoms of asphyxia appear.

CHRONIC CATARRHAL BRONCHITIS.

Etiology.—Chronic catarrhal bronchitis may occur after repeated acute attacks, but it is not common except in rheumatic and gouty patients. It is most commonly secondary to emphysema, to any chronic inflammation of the lung, to pleural adhesions, and to organic heart disease. It usually occurs in elderly people; it is more common in the winter months.

Pathology.—The mucous membrane of the bronchi may be thinned, and the muscular and glandular coats may be either atrophied or thickened and granular. There may be superficial ulcerations. Bronchial dilatation is not uncommon. Emphysema is usually present.

The **symptoms** persist for years—better in summer, worse in winter. There is a cough which is worse at night. The expectoration varies greatly. In some cases it is scanty—the so-called "dry catarrh," an obstinate form with severe and paroxysmal cough. The term "bronchorrhœa" is applied to cases with excessive secretion. The expectoration may be thick and yellowish or greenish, or more watery in character, depending upon the relative proportions of pus, mucus, and serum. If bronchiectatic cavities are present, the patient will often raise large quantities of secretion by any change of position which allows the cavity to drain itself. Dyspnœa is not marked except from associated emphysema or cardiac disease, although some patients develop asthmatic breathing from time to time. There may be mild constitutional symptoms, especially during the winter months—emaciation, slight afternoon rise of temperature, and loss of strength.

Fetid bronchitis occurs when the secretions decompose, especially in bronchiectatic cavities, in tubercular cavities, with abscess and gangrene of the lung, and in empyema with a pulmonary fistula.

Fetid expectoration may occur in chronic bronchitis; it is likely to lead to bronchiectasis, pneumonia, or gangrene of the lung. In these cases it is often difficult to say which is the primary disease.

The **physical signs** are those of bronchitis—coarse and subcrepitant râles, with sibilant and sonorous breathing. Bronchiectatic cavities yield their regular physical signs. If the bronchitis be secondary to pulmonary or cardiac disease, the physical signs of such disease are present.

The **prognosis** is bad for complete recovery, although patients live for years in comparative comfort.

Treatment.—The best possible treatment is to send the patient for the winter to some warm, equable climate. In every case the primary disease should be treated, gouty and rheumatic habits corrected, the bowels regulated, the diet supervised, and every attention paid to the general nutrition. The ordinary expectorants are not of much service. Iodide of potassium in 5- or 10-grain doses three times a day often has a certain curative influence; it should always be tried. Ammonium chloride in 10- or 15-grain doses every two or three hours does good when the secretion is abundant. If there be profuse purulent secretion, turpentine, terpin and terpin hydrate, cubebs, and oil of sandalwood should be tried. For distressing cough the syrup of tar, with or without the syrup of wild cherry or the fluid extract of chekan, may be used. The latter is to be given in ʒj doses every three hours, and the most desirable way of administering it is to evaporate it to a solid extract, which can be given in capsule, after the common method of administering Warburg's tincture.

If fetid bronchitis occurs, myrtol may be used in 2- to

5-grain doses three times a day. Oil of sandalwood and terpin hydrate are also of service, while the odor may be lessened by means of sprays of carbolic acid or of thymol.

CHRONIC CROUPOUS BRONCHITIS.

Etiology and Synonyms.—Nothing definite is known about this rare disease, except that it is more common in adults and in males, and that it is often associated with phthisis. *Synonyms:* Membranous bronchitis; Fibrinous bronchitis; Plastic bronchitis.

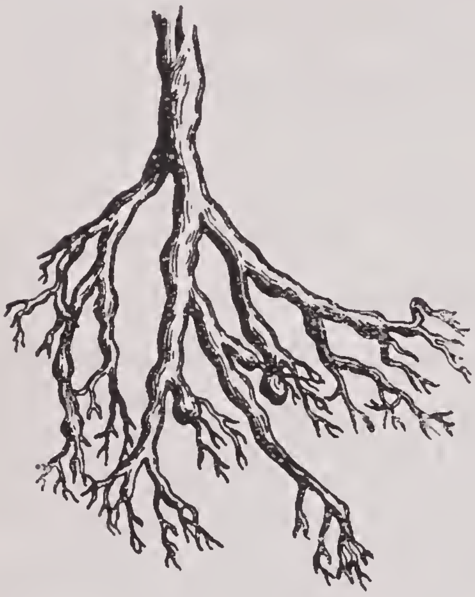


FIG. 23.—Fibrinous bronchial cast.

The **symptoms** of chronic bronchitis are present, and from time to time the patient has a severe coughing attack, possibly with spitting of blood, and raises a cast of a bronchus and its branches. These casts consist of an unknown albuminoid substance, probably of altered fibrin. The attacks occur at varying intervals extending over years, as the disease is essentially chronic.

The **prognosis** for life is good; for recovery from the bronchitis, bad.

The **treatment** is that of chronic bronchitis.

BRONCHIECTASIS.

Definition.—Bronchietasis is a dilatation of the bronchial tubes.

Etiology.—The disease is always secondary to some lesion weaking the bronchial wall, so that it dilates under coughing pressure.

1. There may be congenital weakness of the bronchial wall, usually unilateral, but these cases are extremely rare.

2. Inflammation of the bronchial wall leading to atrophic changes in the muscular and fibrous structures is the operative cause in the large majority of cases; hence the disease occurs with chronic bronchitis, emphysema, broncho-pneumonia of children, phthisis, foreign bodies within the bronchi, or pressure from an aneurysm or a tumor.

3. The bronchial wall may be weakened by traction from without, from old pleuritic adhesions, interstitial pneumonia, and fibroid phthisis.

Pathology.—A *cylindrical* and a *sacculated* form of bronchiectasis are recognized. The two forms may coexist in the same lung. The dilatation varies in size from a pea to that of a small orange. Sacculated dilatations are usually multiple, being spread along the course of a bronchus. A single sacculated bronchiectasis surrounded by non-indurated lung-tissue may occur with emphysema and bronchitis in rare instances, and may resemble a single cyst without contents. The bronchial wall is thinned and its constituent elements are atrophied. The mucous membrane constituting the lining of the cavity may be normal or smooth and glistening, the columnar having been replaced by pavement epithelium, or it may be infiltrated and thickened, or it may be extensively ulcerated, especially in cases where the secretions are retained. The contents of some of the larger cavities are often exceedingly fetid, and a general fetid bronchitis may complicate the disease.

Symptoms.—Moderate bronchiectasis does not, as a rule, give rise to symptoms or physical signs, and it cannot be recognized during life. The larger dilatations are diagnosed by the cough and the expectoration. After a period of some hours free from cough a paroxysm will occur, during which large quantities of sputum are raised, frequently "by mouthfuls." These coughing attacks usually occur in the morning upon arising; they may be brought on by any position of the body that allows the secretion to flow from the dilatation into a normal tube. The sputum is abundant, frequently is foul-smelling, and separates on standing into three layers—the upper of a brownish froth, the middle of watery mucoid substance, and the lowest of a thick sediment of granular matter and cells. When ulceration occurs there may be present hæmatoidin-crystals and elastic fibres. Hemorrhage may occur, but it is rare.

The **physical signs** are not pronounced except in the well-marked cases. Large saccular "dilatations" give rise to the physical signs of cavities, associated with the evidences of the disease to which the bronchiectasis is secondary. The caverous signs vary from time to time according to the amount of accumulated secretion; these signs are often localized at the base of the lung—a point of considerable importance in the diagnosis between this lesion and a tubercular cavity. The physical signs may closely resemble a sacculated empyema with an opening into a bronchus, but the history of the case will usually make the diagnosis evident.

Prognosis.—The condition is essentially chronic, often lasting for years, during which time the patient may enjoy an active life. The prognosis is rendered worse by the primary lung conditions, by hypertrophy and dilatation of the right ventricle secondary to interstitial pneumonia, and by the possibility of an added tubercular infection or of pulmonary gangrene.

Treatment is unsatisfactory, because it is inadequate to heal the dilatation. The cough is beneficial in clearing out the accumulated secretions, hence narcotics are inadmissible. Stimulant expectorants are useless. Some benefit may result from the administration of terpin hydrate in full doses, terebene, and turpentine. Injection of antiseptic solutions into the cavities has been followed by good results in some cases. In patients in good condition, with superficial cavities, incision and drainage may be resorted to. For the fever myrtol may be given internally, and inhalation of carbolic acid (1 to 3 per cent. solution) and of thymol (1 : 1000), as described under Fetid Bronchitis; the results, however, are never very satisfactory in extreme cases.

ASTHMA.

Definition.—Asthma is an affection characterized by paroxysmal dyspnœa due to contraction of the bronchi. The same name also designates the paroxysmal dyspnœa, due to the contraction of the arteries, commonly seen with emphysema.

Etiology.—A number of theories have been advanced to explain the symptoms: (1) That the disease is due to spasm of the muscular tissue of the small bronchi (Biermer). (2) That it is due to spasm of the diaphragm and the accessory muscles of respiration (Wintrich-Bamberger). (3) That it is a vaso-motor neurosis causing sudden swelling of the bronchial mucous membrane (Störck, Sir Andrew Clark, Traube). (4) That it is due to the elimination of Leyden's crystals. (5) That it is a special form of inflammation of the bronchioles—bronchiolitis exudativa (Curschmann). Of these theories, the first, that of bronchial spasm, is the one generally adopted.

The **causes** of the disease are both predisposing and exciting.

Predisposing Causes.—The disease is more common in those with high-strung nervous systems; it may run in neurotic families associated with epilepsy or with neuralgia. Males are more frequently affected than females. As to age, of 225 cases, 71 occurred in the first decade, 30 in the second, 39 in the third, 44 in the fourth, 24 in the fifth, 12 in the sixth, 4 in the seventh, and 1 in the eighth. The predisposition to the disease is frequently retained throughout life. The affection is more common in those with pulmonary emphysema.

Exciting Causes.—Climatic influences are very curious, some patients having asthma in some places and not in others, without apparent reason. Vegetable and terrestrial dust and irritating vapors may induce an attack. In this respect asthma closely resembles hay fever, with which disease it is closely allied, and which it frequently complicates. Ipecac, sulphur, iodine, the pollen of many flowers and grasses, the irritant odor of violets, roses, and strawberries, the dust of feathers, and the emanations of certain animals afford familiar examples of personal susceptibility. Sudden mental shocks and deep emotions may induce asthma. The most frequent cause of an attack in those predisposed to asthma is bronchitis, and if in such patients bronchitis can be avoided, attacks of asthma are rare.

Reflex causes are common. Nasal polypi, hypertrophic rhinitis, naso-pharyngeal adenoids, and enlarged tonsils are frequently found, and the cure of these conditions will in many cases remove the liability to asthma. It is too much to claim that nasal and pharyngeal lesions comprise the only cause, however, as has been done by some. Among other reflex causes are uterine and ovarian diseases, overloading of the stomach, and the taking of certain articles of food.

Pathology.—As asthma is a functional disease, there is no regular lesion, although in old asthmatics emphysema and chronic bronchitis are often present.

Symptoms.—There may be premonitions—a sense of drowsiness, depression of the mind, tightness in the chest, or peculiar feelings in individual cases that mean an impending attack. The paroxysm usually begins at night with a sense of dyspnœa and with laborious efforts at breathing. The patient cannot lie down, but sits or stands, usually by an open window. Inspiration is spasmodic; expiration is prolonged and wheezing. The accessory muscles of respiration are called into play; the face is livid and distressed, and perhaps cyanotic. Limpid urine is usually passed in large quantities. There is a cough, tight at first, with ball-like gelatinous masses of sputum—the "*perles*" of Laennec. These balls can be unrolled in water; they represent mucous casts of the smaller bronchi. They frequently have a distinct spiral form, and they are known as "Curschmann's spirals" (Fig. 24), in which there is frequently a central translucent filament composed of altered mucin. In addition are found in the sputum the pointed octahedral crystals described by Leyden, identical with those found in semen and in leukæmic blood (Fig. 25).

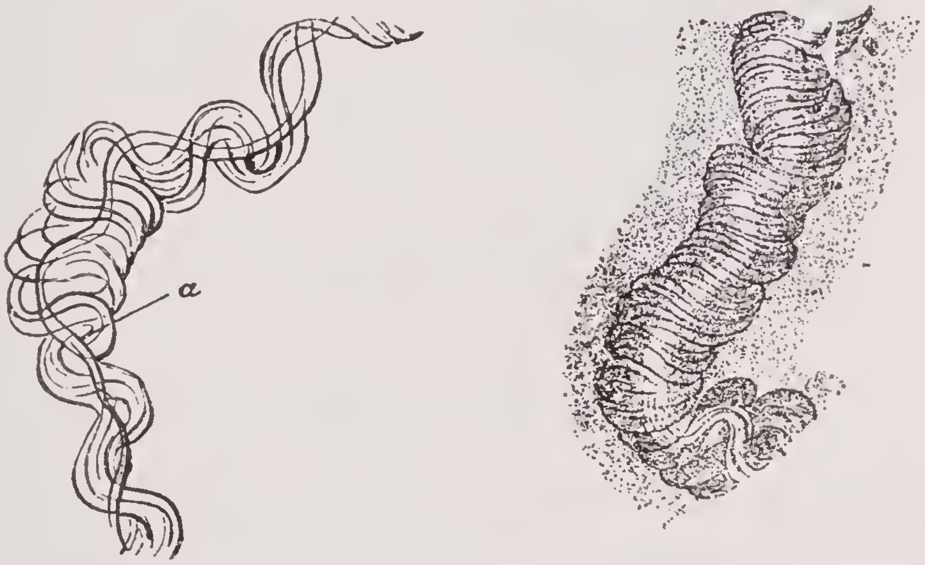


FIG. 24.—Curschmann's spirals: *a*, central fibre (after Curschmann).

Physical Signs.—The chest is fixed and enlarged—often from 6 to 8 centimeters larger in circumference than normal. Expansion is poor, especially laterally, and is in strong contrast to the muscular attempts of respiration. Inspiration is short; expiration is prolonged and wheezing.

The diaphragm is low and moves but slightly. Percussion shows an increased area of pulmonary resonance. The note may be normal or hyper-resonant or tympanitic. On auscultation are heard all varieties of sibilant, sonorous, cooing, and whistling râles, especially during expiration.

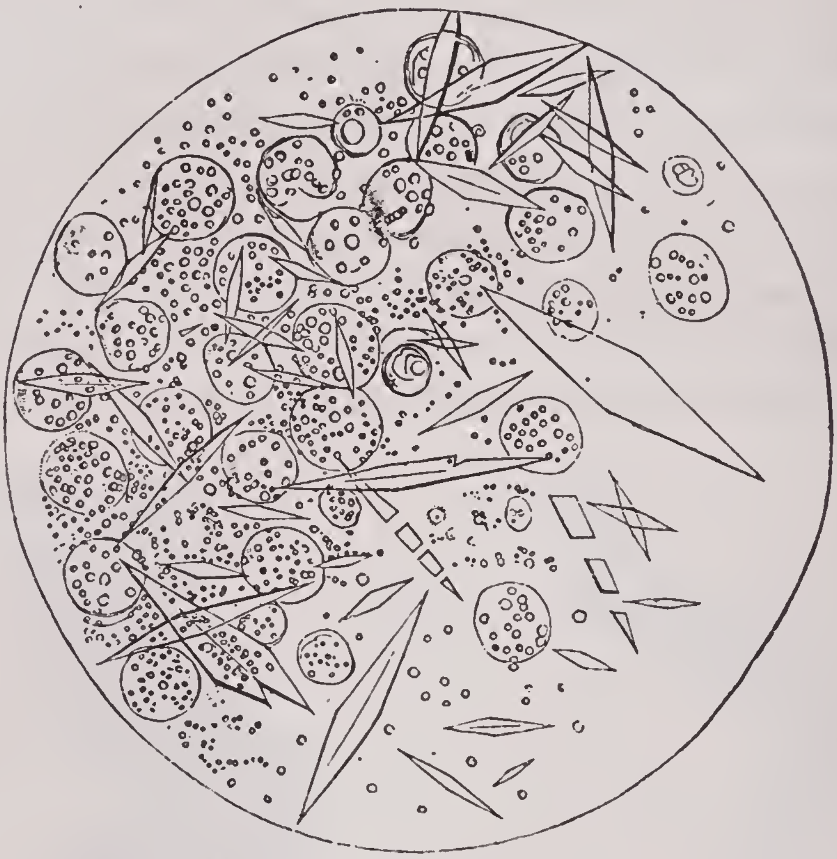


FIG. 25.—Charcot-Leyden's asthma-crystals (after Riegel).

The expiratory murmur may be prolonged, or breathing sounds may be absent or be obscured by the râles.

The **duration** of the attack varies from several hours to a number of days. In the more protracted cases the symptoms are worse at night. Between the attacks there may be dyspnœa, wheezing respiration, and cough. In the long-standing cases emphysema and chronic bronchitis develop, resulting in chronic invalidism.

The **prognosis** for life is good, death never resulting during a paroxysm.

Treatment.—*During the attack* immediate treatment is required to relax the contracted bronchioles. A number of remedies may be employed, Of these remedies amyl nitrite is the most serviceable, a *perle* containing from 2 to 5 minims being broken in a handkerchief and the vapor inhaled. Hot stimulants or spirits of chloroform in hot water may be given, while whiffs of chloroform may be required in aggravated cases. Permanent relief is often afforded, even in obstinate cases, by a hypodermic injection of morphine. Nitroglycerin, gr. $\frac{1}{100}$ every two or three hours, is of service in the more protracted cases. Choral in 10- or 15-grain doses often affords relief. Antipyrine, gr. xv, or phenacetine, gr. x, may be used, repeated every three hours. Good results are claimed for the fluid extract of grindelia robusta in ʒj doses every four hours. The smoke of cigarettes containing hyoscyamus, belladonna, or stramonium may be inhaled, or pastilles may be made from these drugs, with the addition of potassium chlorate or nitrate. Inhalations of cigar-smoke are frequently of great value. Paper saturated with a strong solution of potassium nitrate burnt in the room before retiring will often ward off a nocturnal attack.

Between attacks antispasmodics should be given. Iodide of potassium in gr. v–xv doses three times a day, with or without the addition of 5 grains of chloral to each dose, is of great benefit. Nitroglycerin, gr. $\frac{1}{100}$ every four to six hours, may be used. The systematic inhalation of compressed air has been recommended strongly.

The diet should be such as not to induce flatulence, carbohydrates being used in great moderation. The patient should not retire to bed on a full stomach, and it is usually best for the heavy meal of the day to be taken at noon.

If nasal polypi or hypertrophies are present, they should be removed.

For every asthmatic there are localities in which he has little or no asthma. The particular locality of exemption should be found by each patient to suit his individual case, as no regular rule can be laid down to suit all patients.

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3. DISEASES OF THE LUNGS.

(*a*) CIRCULATORY DISTURBANCES.

CONGESTION OF THE LUNGS.

Congestion may be either active or passive.

Active congestion occurs with acute inflammation of the lungs, with over-action of the heart, and from the inhalation of hot or irritating vapors. It may cause increase of dyspnœa, cough, and expectoration, with a moderate degree of fever, but about its symptomatology not much is accurately known.

Most authors describe a rapidly fatal form of congestion occurring after exposure to cold or after over-exertion.

Passive Congestion.—Two distinct forms of passive congestion are recognized—mechanical and hypostatic.

1. *Mechanical congestion* is known as "brown induration" or the "pneumonia of heart disease"; it is described under the heading of Chronic Venous Congestions of Heart Disease, page 209.

2. *Hypostatic congestion* of the posterior portions of the lungs is often found as the result of post-mortem changes. It is common in those confined to bed for a long time in a weakly condition, as the combined result of feeble circulation and the effect of gravity. In coma and in cerebral injuries, such as cerebral hemorrhage, it is often seen in its most pronounced degree. The affected portions of lung are congested, œdematous, heavy, and imperfectly aërated.

The congestion may be complicated by patches of consolidation resembling either broncho-pneumonia or a lobar pneumonia, being due to the passage into the bronchi of food or air containing streptococci.

The **symptoms** are usually obscured by those of the primary disease, so that a diagnosis is to be made by physical signs. There is dulness over the congested portions, with feeble breathing and liquid râles. In more advanced cases there may be bronchial breathing and bronchophony.

ŒDEMA OF THE LUNGS.

Localized œdema of the lungs occurs with congestion, inflammation, and new growths; it is known as "collateral œdema."

General pulmonary œdema occurs from weakness of the left side of the heart, the force of the right heart being unimpaired, or it may occur in pronounced conditions of hydræmia. Either cause alone may suffice for its production, although in extreme cases both factors are usually present. It is often present during the death agony, being a symptom of approaching death. It is seen in the final stages of cachexias, profound anæmia, acute and chronic Bright's disease, pneumonia, cerebral diseases, and diseases of the heart.

The lungs are heavy, pit on pressure, and from their cut section exudes a frothy serous or sero-sanguinolent fluid in abundance. This fluid is also present in the trachea and the bronchi. The œdema interferes with the proper degree of aëration, although cut pieces of the lung still float in water. There is usually associated congestion of the lungs, especially of the posterior portions.

The **symptoms** are increasing frequency of respirations, dyspnœa with cough, and the expectoration of serum which may be blood-tinged. The respirations are bubbling and rattling, and cyanosis, increasing coma, and cold, clammy extremities precede the fatal issue. The percussion-note over the œdematous portions is more or less dull; the respiratory murmur is feeble or is obscured by large liquid râles and bubbling sounds usually first heard at the bases.

The **treatment** is that of the primary condition. The heart should be stimulated energetically; cups and poultices are to be applied to the chest, and the bowels should be moved freely. Venesection should be resorted to in acute cases with cyanosis; it frequently affords relief.

PULMONARY HEMORRHAGE.

Two forms of pulmonary hemorrhage are recognized: 1. Broncho-pulmonary hemorrhage, or bronchorrhagia, in which condition the blood is poured into the bronchi and is expectorated; 2. Pulmonary apoplexy, or pneumorrhagia, in which disease the hemorrhage occurs into the substance of the lung.

Broncho-Pulmonary Hemorrhage, or Hæmoptysis.

There is a variety of causes giving rise to this condition.

(*a*) Pulmonary tuberculosis is the most common cause, and it should always be suspected, even if neither symptoms nor physical signs be present. Small repeated hemorrhages in the earlier stages are due to bronchial congestion or ulceration. Large hemorrhages in the later stages arise in cavities from erosion of a branch of the pulmonary artery or from rupture of an aneurysmal dilatation of the same.

(*b*) Hemorrhages may occur in young people without assignable cause, although in some cases they may follow excitement or severe muscular exertion, especially in high altitudes.

(*c*) Anæmic hysterical women may raise a little blood from time to time without apparent reason. Deception must always be suspected in such cases, however.

(*d*) Severe injuries and contusions of the chest are often followed by hemorrhage.

(*e*) Patients with emphysema and bronchitis may occasionally raise small quantities of blood.

(*f*) Hemorrhage may result from certain diseases of the lung, the initial stage of pneumonia, cancer, gangrene, abscess, or bronchiectatic cavities.

(*g*) Vicarious hæmoptysis may occur with interrupted menstruation. It has been known to follow removal of both ovaries.

(*h*) Small repeated hemorrhages are common with the pulmonary congestion of heart disease, especially with lesions of the mitral valve.

(*i*) With aneurysms small quantities of blood may be raised from congestion of the bronchi from pressure or by leakage through a small perforation. Large and fatal hemorrhage results from rupture of the sac into the trachea or into a bronchus.

(*j*) Any ulcerative process in the larynx, the trachea, or the bronchus may cause small repeated hemorrhages.

(*k*) Sir Andrew Clark describes a form of hæmoptysis, seen in elderly people, which he calls "arthritic hæmoptysis." It occurs in those of the arthritic diathesis; it is due to minute structural alterations in the terminal blood-vessels of the lung. The prognosis in these cases is usually good.

(*l*) Hæmoptysis occurs with extensive blood-alterations of malignant fevers, as hemorrhagic variola, and with purpura hæmorrhagica.

(*m*) In Japan and China occurs an endemic hæmoptysis due to the presence of the *Distoma Ringeri* in the bronchi.

Symptoms.—There may be a preceding feeling of oppression in the chest, but usually the first symptoms are a warm, mawkish taste in the mouth, nausea and faintness, and the appearance of the blood. The quantity of blood ejected varies from a dram to a pint or more.

Anæmic symptoms—faintness, syncope, dyspnœa, "air-hunger," and pallor—depend on the quantity of blood lost. Large hemorrhages may be fatal from anæmia or from the filling of the bronchi with blood, but usually danger is not imminent. There is generally but little effort in raising the blood. Should the quantity be large, a certain amount may be swallowed, to be vomited later or passed with the stools. Blood from the lungs has certain characteristics which distinguish it from blood from the stomach. It is scarlet in color, of an alkaline reaction, frothy, and mixed with mucus. In the clots air-bubbles can usually be seen. After a hemorrhage the sputa are usually blood-stained, of a dark crimson or brown color, and frequently clots like bronchial casts are raised. Vomited blood is dark brownish, contains no air, but is mixed with stomach-contents and is of an acid reaction. Blood coming from the pharynx or the nares is usually hawked up, and on inspection blood-streaks can almost always be seen descending from the naso-pharynx.

Large hemorrhages, leading even to a fatal issue, can take place into extensive pulmonary cavities without blood being coughed up at all. The hemorrhage may continue for several hours or even days, and attacks may be repeated from time to time. They may be induced by exertion, by over-indulgence in stimulants, or by excitement, but in some cases they occur without apparent cause, even while the patient is resting quietly at night.

Anæmic symptoms follow large hemorrhages, but after small hemoptyses the patients frequently feel much relieved in their pulmonary or cardiac symptoms.

Treatment.—For the large hemorrhages arising from erosion of an artery or from rupture of an aneurysm treatment is unavailing. The patient should be kept absolutely quiet and secluded, and small doses of opium should be given to relieve the restlessness and steady the heart. Fainting is nature's measure of tranquillizing the circulation and inducing firm thrombosis.

Anæmic symptoms are treated by elevation of the foot of the bed, by ligating the extremities, and by hypodermic injections (hypodermoclysis) or arterial transfusion of sterilized saline solutions, which may also be given by the rectum. Internal hæmostatics are useless.

In less serious hemorrhages the patient may be given ice to swallow and may drink small quantities of aromatic sulphuric acid in water. Theoretically, measures to reduce the frequency of the heart-beats and reduce the blood-pressure in the pulmonary circulation are indicated, but our knowledge as to how this latter indication can be fulfilled is very meagre. Rest should be enforced, and opium be given to quiet the patient. The diet should be light, and stimulants should not be employed. Digitalis is contraindicated. Aconite may be given with benefit if there be vascular excitement. Acid drinks and cracked ice may be given. Ergot, gallic acid, acetate of lead, hydrastis, and krameria are used as routine measures, but are of doubtful utility. Ice applied to the chest is recommended, and in some cases it seems to do good. Free purgation is indicated to reduce blood-pressure; it should be resorted to in all protracted cases.

PULMONARY APOPLEXY; HEMORRHAGIC INFARCT.

Hemorrhage into the substance of the lung, with rupture of its tissue, may occur with severe contusions, with penetrating wounds, and with rupture of an aneurysm. Aside from these cases, so-called "hemorrhagic infarcts" result from embolism or thombosis of a branch of the pulmonary artery, resulting in the stoppage of its circulation. As these are "terminal" arteries, without anastomotic branches, the blood in the vessels beyond the obstruction is in a condition of stasis, and congestion occurs from a backward pressure into the shut-off region. The vascular walls lose their consistency and allow the escape of blood into the surrounding structures.

The hemorrhagic area is red and solid, resembling a blood-clot, becoming reddish-brown in time from pigment-changes. It is of a wedge shape with the base out; the pleura covering it is usually inflamed. It is usually situated toward the base, and it varies in size from a walnut to that of an orange. There may be a surrounding zone of pneumonia. Such an infarction may be absorbed if small, but it usually becomes changed to a pigmented, puckered cicatrix. In rarer cases it may undergo sloughing or gangrene. Abscess results if the cause be an infectious embolus containing suppurative micrococci, as may occur in pyæmia or in malignant endocarditis.

Symptoms.—There is usually pain in the side, sudden dyspnœa, oppression in the chest, and bloody expectoration, in some cases amounting to a fair-sized hæmoptysis. Physical examination yields over the hemorrhagic area, if it be of sufficient size, dulness, bronchial voice and breathing, and pleuritic and bronchial râles. Large hemorrhagic infarcts may be followed by sudden death. It must be remembered that obstruction of a large branch of the pulmonary artery can occur without the formation of a hemorrhagic infarct.

The **treatment** is practically that of pneumonia.

LOBAR PNEUMONIA.

Definition and Synonyms.—Lobar pneumonia is an infectious disease due to the diplococcus pneumoniæ; it is characterized by an inflammation of the lung with constitutional symptoms. *Synonyms:* Croupous or Fibrinous Pneumonia; Pneumonitis.

Etiology.—The diplococcus pneumoniæ of Fränkel, commonly known as the "pneumococcus," is the specific germ of the disease. It is an ovoid coccus, or, more properly speaking, a bacillus, usually occurring in pairs, and more often encapsulated. It is found in the nasal and buccal secretions of 20 per cent. of healthy people, and after an attack of pneumonia it is often found in the mouth for months; hence there must be conditions giving the germ at times more intense pathogenic properties, or conditions rendering the individual susceptible to the infection. Among these causes may be mentioned exposure to hardship and cold; consequently the disease is more frequent in men than in women. It frequently follows immersion in cold water. Traumatisms of the chest-wall produce the so-called "contusion-pneumonia." Alcoholism, chronic Bright's disease, and any condition of bodily weakness predispose to the disease. Repeated attacks may occur; they are explained by auto-infection from the persistence of diplococci in the buccal and nasal secretions. The disease is more common in the months from February to May, although it may occur at any time. It occurs in all temperate climates, but it is unknown north of Labrador. It is more frequent in

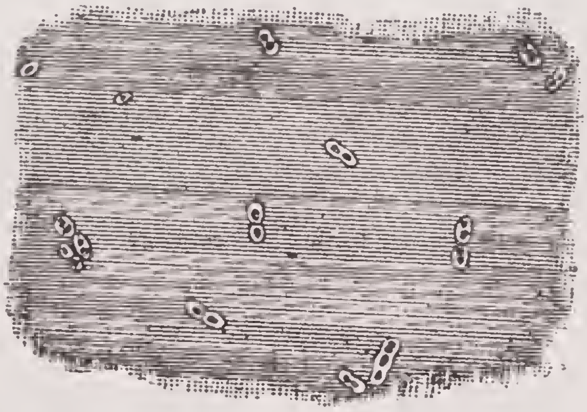


FIG. 26.—Fränkel's pneumonia coccus, bred from the expectoration. (Prepared by Prof. Gärtner. Oil-immersion lens $\frac{1}{12}$; eye-piece No. 4.)

the Southern than in the Northern States. The influence of age is important. Liability to the disease increases up to the twentieth year, then decreases until liability is again increased in old age. Children under five years of age usually have broncho-pneumonia; those between five and fifteen years of age have either lobar pneumonia or broncho-pneumonia; adults usually have lobar pneumonia.

The diplococci are found not only in the exudate in the inflamed lung, but also in many of the complicating lesions, as in the meninges, the pleura, or the pericardium; they may even involve these parts without there being any inflammation of the lung at all.

Pathology.—The lesion involves a whole lobe, a part of a lobe, or the entire lung. The lower lobes are more frequently involved than the upper; the right lung is more often involved than the left. The process is divided into four stages: congestion, red hepatization, gray hepatization, and resolution.

Congestion.—The lung is congested and heavy; its cut surface is bathed with bloody serum. Microscopic examination shows congestion of the blood-vessels and swelling and proliferation of the alveolar epithelium, while the air-cells are partially filled by an exudate of fibrin pus-cells, red blood-corpuscles, and epithelial cells. The stage of congestion usually lasts for several hours, but it may be protracted for several days.

Red Hepatization.—The lung is large, often showing indentations of the ribs, and is remarkably friable. It is hard and airless, cut pieces sinking in water. There is fibrin on the pulmonary pleura. The cut surface is dry, reddish, and distinctly granular, due to the protrusion of inflammatory exudate from the air-cells. The microscope shows the air-cells, and frequently the small bronchi as well, filled with an exudate of fibrin, pus-cells, red blood-cells, and epithelium.

The blood-vessels are congested but pervious. The interstitial connective tissue of the lung may be infiltrated with inflammatory exudate. Diplococci, and occasionally staphylococci and streptococci as well, are seen in the exudate. One-fourth of the fatal cases occur in red hepatization from the first to the eleventh day of the disease.

Gray Hepatization.—The color changes from reddish-brown to gray, at first in spots, so that the lung has a mottled appearance. The lung still remains solid, but the exudate is decolorized and begins to soften and degenerate. One-half the fatal cases occur in the mottled condition, between the second and the eighteenth day, and one-fourth in the completed gray stage, between the fourth and the twenty-fifth day.

Resolution occurs in spots at first. Fatty degeneration and liquefaction of the exudate occur, allowing of its absorption and expectoration. Resolution should begin soon after defervescence, but it may be delayed.

The unaffected portion of the lung may be congested and œdematous, especially the portions near the affected area. The bronchi of the pneumonic lobe show catarrhal inflammation, and they may be filled by fibrinous plugs.

Modifications of the Lesion.—Resolution may be delayed, the lung remaining in dry gray hepatization for weeks. The exudate may be so excessive that the blood-vessels are compressed, leading to necrotic changes of portions of the lungs. There may be an excessive production of pus-cells, which infiltrate the connective-tissue septa, forming small abscesses or a diffused suppuration. Gangrene of the lung may occur. Instead of resolution the exudation may become organized into connective tissue, so that the air-cells are obliterated and rendered permanently unfit for use. The bronchitis may be general and excessive, especially in cases accompanying epidemic influenza.

Complicating lesions result from infection of other parts by diploccoci. Pleurisy is only to be considered a complication if it extend beyond the pneumonic area or if serous or purulent effusion occur.

Pericarditis is not infrequent. It occurs more commonly with double or left-lung pneumonias and in children. It is due to diplococcus infection, producing plastic inflammation or serous or purulent effusion. The prognosis of pleurisy and pericarditis of pneumonic origin is better than that of the other forms.

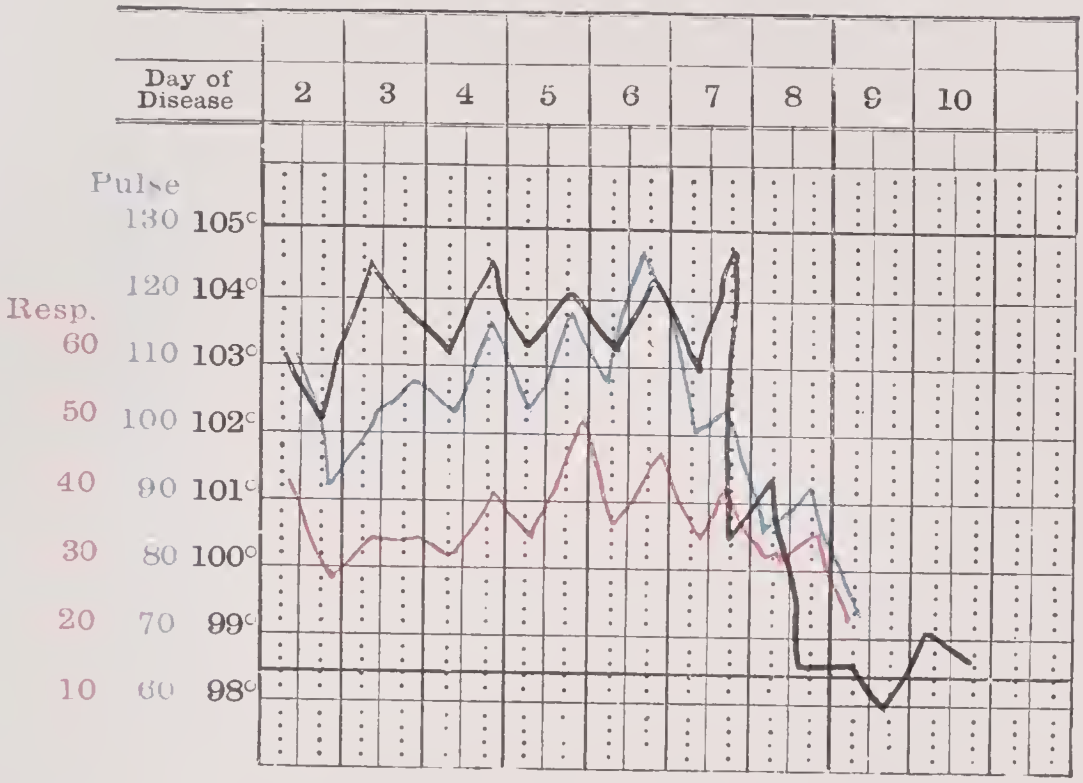
Endocarditis is more common than pericarditis. It may be either simple or malignant; it occurs as an acute exacerbation not infrequently in those who have old valvular disease.

Meningitis may occur, and it is usually associated with malignant endocarditis. It is often difficult to tell whether pneumonia or meningitis be the primary disease. Croupous gastritis or colitis may occur. The liver and the kidney usually show parenchymatous changes.

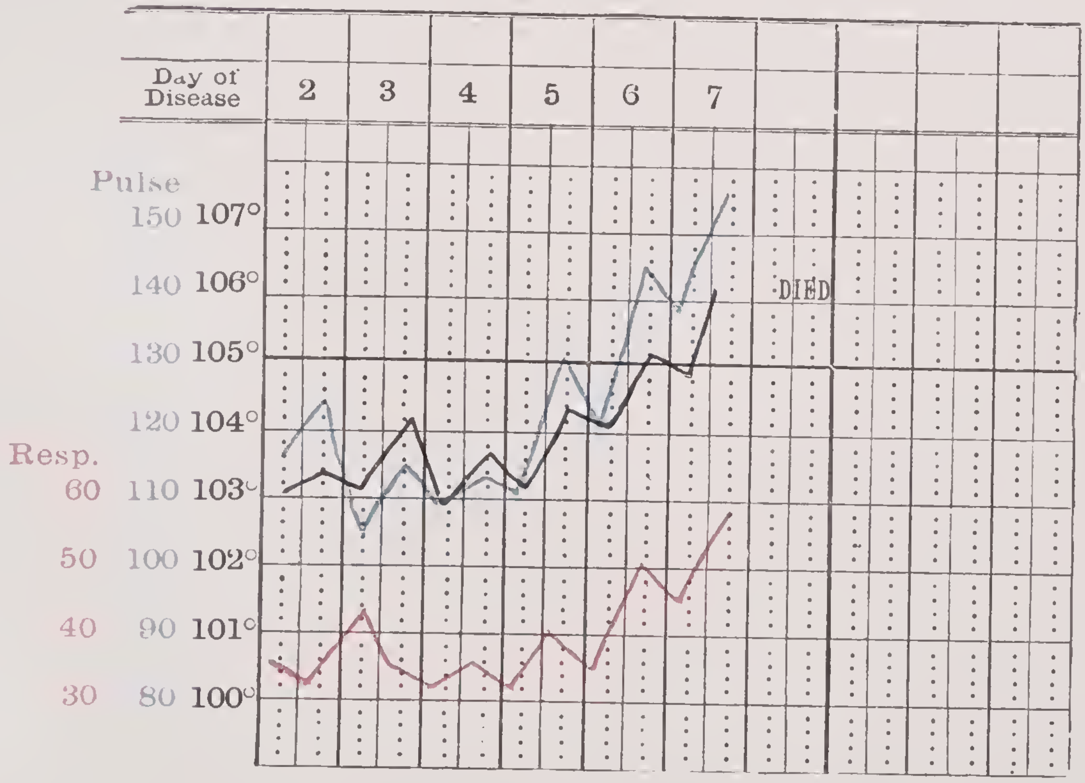
Symptoms.—*Prodromal symptoms*, consisting of malaise, dull pain in the back and the bones, and some soreness in the chest, are present in about one-fourth of the cases. They last for a day or so, and probably are due to a protracted stage of congestion.

The actual onset of the disease is marked by one or more *chills* in about 90 per cent. of the cases, and from the chill the duration of the disease is reckoned. In children convulsions or vomiting may replace the chill. In old people a shivering attack and pain in the side may be the only symptoms.

The *temperature* rises rapidly and attains its maximum in from twenty-four to thirty-six hours, although in some cases the height of the fever is not reached until the day before defervescence. The fever remains high with slight evening



Lobar pneumonia terminating by crisis on the seventh day: temperature (black), pulse (blue), and respiration (red).



Temperature chart of a fatal case of pneumonia: temperature (black), pulse (blue), and respiration (red).

exacerbations, which in uncomplicated cases should not exceed 104° F.

Defervescence may occur at any time between the second and the eighteenth day, the seventh, fifth, eighth, sixth, and ninth days being the favorites, in the order named. The temperature may fall in from six to forty-eight hours by crisis, or in from three to five days by lysis. In some cases the

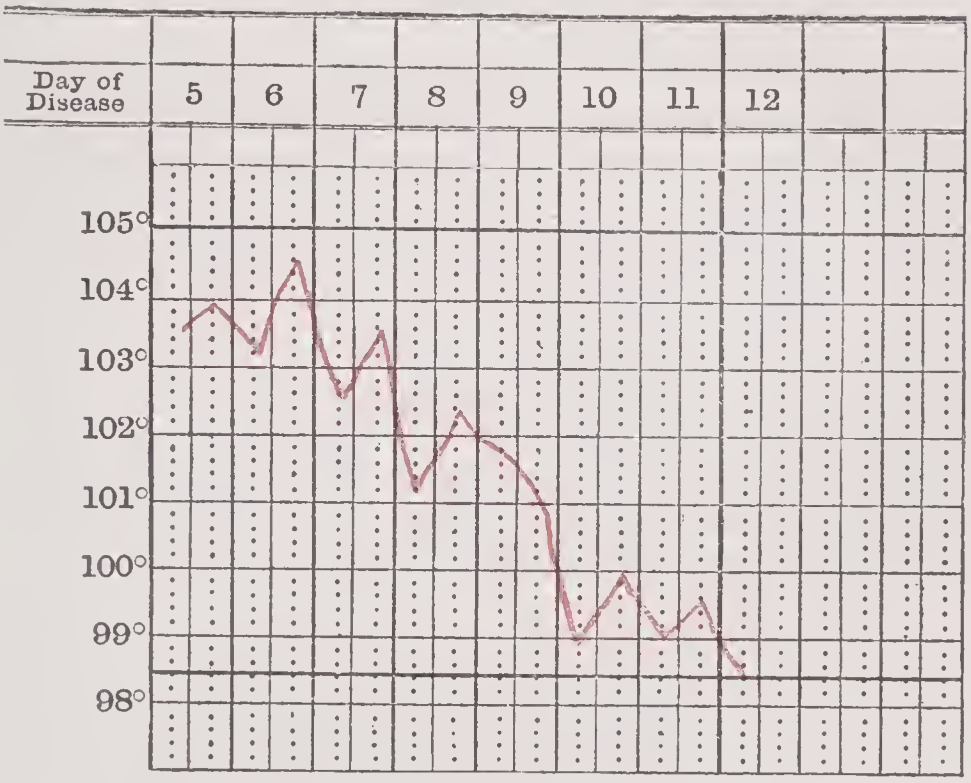


FIG. 27.—Temperature chart of pneumonia terminating by lysis.

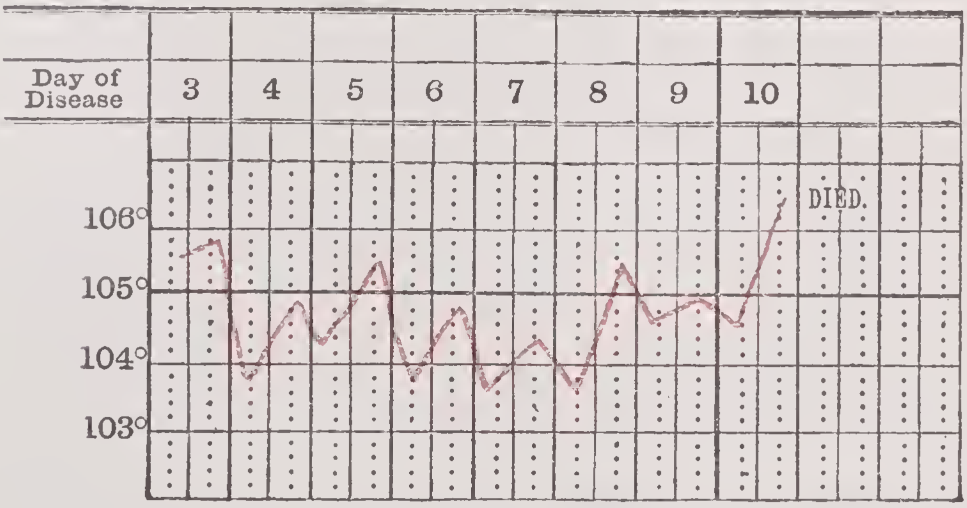


FIG. 28.—Temperature chart of pneumonia with purulent inflammation terminating fatally.

temperature is markedly remittent at any time in its course, especially in children. In other cases a pseudo-crisis occurs about the fifth day. At the time of the crisis the temperature may fall to subnormal, and after the crisis a slight rise of fever is noticeable for two or three days, especially in the evening. A high temperature persisting for ten days suggests purulent infiltration or empyema. A sudden rise of temperature at any time indicates a complication or an extension of the disease. In old people the temperature may be normal or even subnormal. In the pneumonias complicating epidemic influenza the temperature does not

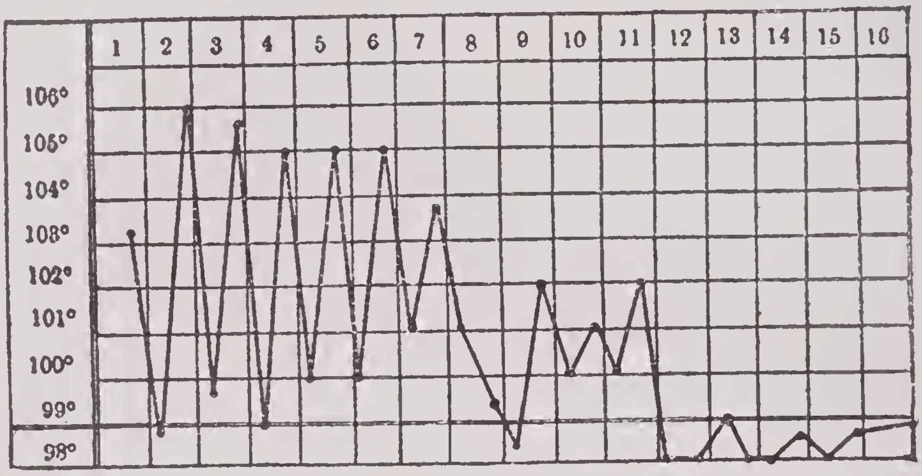


FIG. 29.—Lobar pneumonia in child, with remittent temperature (Holt).

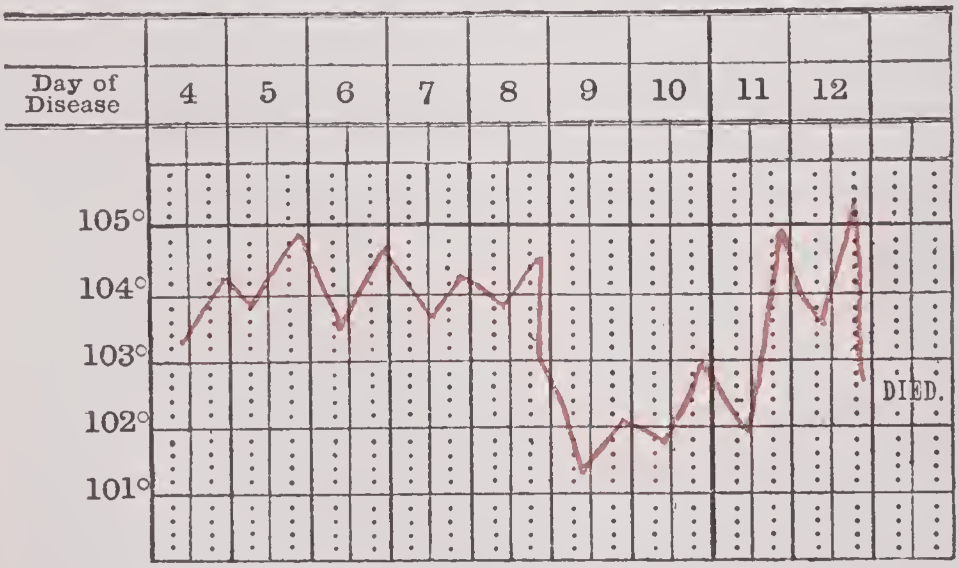


FIG. 30.—Lobar pneumonia; defervescence; gangrene of lung.

become normal for days, and it may even persist after resolution.

The character of the heart's action is of the utmost importance, as heart failure constitutes the greatest danger of pneumonia. The pulse should be full and about 100 in a favorable case. A pulse over 120 passes the safety limit and gives cause for anxiety. The most critical time is just before defervescence, at which time liability to sudden or gradual heart failure is the greatest. The possibility of sudden death must always be considered.

In old people rigidity of the arterial walls may give a fictitious tension to the pulse, and it is best to note the character of the action of the heart itself. Of equal importance with a rapid pulse in old people is an irregular and intermittent heart-action.

A fall in the pulse even to 50 may be noticed before crisis. In case of rapid defervescence there may be extreme prostration and heart failure. A pulse running up rapidly to 140 indicates paresis of the medulla and often precedes the fatal issue. In children, however, a rapid pulse is not of so much importance, recovery being possible even with a pulse of 150 to 200.

The *breathing* is rapid and oppressed. Its rapidity varies with the amount of fever, the extent of lung involved, the severity of pleuritic pain, and the presence of complications. The respirations should be below 40 to the minute, a temperature of 104° F., respiration of 40, and a pulse of 120 being the safety limits. If the respirations be over this limit, there should be suspicion of pleuritic effusion or pulmonary œdema.

Dyspnœa may be marked in some cases, especially in upper-lobe pneumonias. It is not usually noticed in old people, in whom the respiration may not even be increased.

Pain in the chest, usually referred to the nipple, occurs in 85 per cent. of all cases. It is a true pleuritic pain, and it does not occur unless the pleura is inflamed. It is a fairly constant symptom in old people.

Cough usually comes early in the disease, but it may be deferred until after defervescence, or it may be absent altogether, especially in old people.

The *sputum* is scanty, gelatinous, and rusty in color, the little pellets sticking to the side of the cup; it is usually mixed with the ordinary sputum of bronchitis. This " rusty sputum " is very characteristic. At the onset there may be expectorated a little pure blood. In bad cases there may be the " prune-juice " expectoration of an abundant dark-brown fluid. This sign is a serious one. In some cases the patient will raise casts of the small bronchi that unroll in water. In children the sputum is usually swallowed instead of being expectorated.

Cerebral Symptoms.—Headache, restlessness, and sleeplessness are frequent; they appear to be due to the pyrexia. *Delirium* belongs to the severe forms of the disease; it is said to be more common in apex pneumonia. In some cases it depends upon the pyrexia and is marked by mental wandering, especially at night. In debilitated subjects and in very severe cases the symptoms of the " typhoid state " appear—rapid feeble pulse, great prostration, brown dry tongue, and a muttering delirium. The last-named symptom is commonly seen in alcoholic subjects, who are, moreover, liable to develop the typical symptoms of acute delirium tremens. Old people are apt to wander in the mind and to evince a constant desire to leave their bed and walk about; as a rule, however, they are readily controlled. In some non-alcoholic adults there may be acute mania. In children cerebral symptoms are more constant, often presenting the clinical picture of acute meningitis. Convulsions at the onset may be followed by headache, photophobia, boring of the head into the pillow, convulsive movements, and coma.

Digestive symptoms are usually not marked. There may be nausea and vomiting, especially in alcoholic subjects, and in some cases a catarrhal jaundice appears. The bowels are usually constipated; the tongue is coated. A brown dry tongue belongs to the most severe cases.

The *urine* is that of fever, concentrated and depositing urates. There is usually a diminished amount of sodium chloride, but this sign is not diagnostic. In about one-third of the cases albumin and casts are present from a complicating parenchymatous nephritis, which, however, gives no symptoms. It is important to examine the urine in every case, to determine the presence of chronic Bright's disease, which adds so grave an element.

The appearance of the patient is often characteristic. The decubitus is usually on the affected side. The expression is both apathetic and anxious. There is frequently a well-defined mahogany-colored flush on one or on both cheeks —not always limited, as was once supposed, to the affected side. Herpes labialis occurs in a certain number of cases: this sign is to be regarded as a favorable one. The herpetic vesicles frequently contain the pneumococci.

The **physical signs** may be described as those occurring (1) in the stage of congestion, (2) in the stage of consolidation, and (3) in the stage of resolution.

1. *Stage of Congestion.*—The normal physical signs are the crepitant râle, the subcrepitant râle, a slight dulness, and a localized deficiency in chest-expansion. The crepitant râle consists of a shower of fine dry crackles, heard directly under the ear at the end of inspiration only. This râle was formerly considered pathognomonic of pneumonia, and to be due to the inspiratory stretching of the alveolar walls stiffened by inflammatory infiltration. It is now con-

sidered to be a pure and simple pleural râle, and is consequently present only when the pleura is inflamed. The subcrepitant râle may arise either from the rubbing together of the inflamed pleuræ or from exudate in the small bronchi. Slight dulness is apparent by a percussion-note of shorter duration, higher pitch, and less resonance than normal. Deficiency in chest-expansion is best appreciated by palpation.

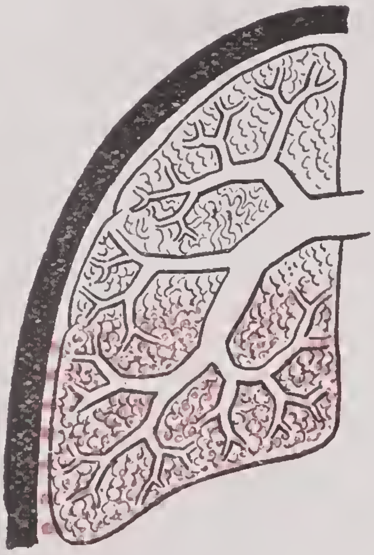


FIG. 31.—Physical signs of lobar pneumonia during the stage of congestion: slight dulness or dull tympany; breathing feeble or harsh; crepitant and subcrepitant pleuritic râles.

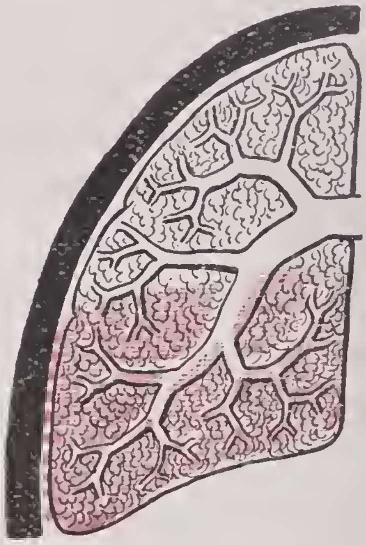


FIG. 32.—Physical signs of lobar pneumonia during the stage of complete consolidation: dulness; bronchial voice and breathing; increased vocal fremitus; crepitant and subcrepitant pleuritic râles.

Exceptional Signs.—There may be a general bronchitis with bronchial râles obscuring the physical signs. In these cases the disease may be mistaken for bronchitis or for tubercular disease. There may be only an area of feeble or harsh breathing, with an occasional subcrepitant râle. Instead of slight dulness there may be a tympanitic note due to relaxation of the congested alveolar walls, or a note of mixed tympany and dulness. Should the lesion begin in the deeper parts of the lung, physical signs may be absent entirely. If the pleura be not involved, the crepitant râle is not present.

2. *Stage of Consolidation.*—The normal signs are dul-

ness, increased vocal fremitus, bronchial voice and breathing, crepitant and subcrepitant râles, and diminished chest-expansion. Dulness is shown by a short, high-pitched note of feeble resonant quality, with an appreciable lack of resiliency to the percussing finger. Bronchial breathing consists of a harsh, loud, and high-pitched respiratory murmur, in which the sound produced by expiration is higher pitched and more prolonged than that of inspiration. Bronchial voice is high-pitched, loud, and nasal in quality.

Exceptional Signs.—The dulness may have a decided tympanitic quality, resembling in some cases even the "cracked-pot" or the amphoric note. These modifications are more commonly observed in pneumonia of the apex; they are especially marked in children. In some cases the note may be flat, especially if the bronchi be filled with exudate, but the flatness is never so complete as in pleural effusion. Should the pneumonic area be small, dulness may be obscured by the tympanitic note produced in the surrounding lung-tissue by its congestion and relaxation.

Bronchial voice and breathing may be absent. They depend upon the flow of air in the bronchi, and they are absent if the bronchi are occluded or if the lung does not expand with respiration; hence they may be brought out by coughing or by deep breathing.

The crepitant râle may be absent, either because the pleura is not involved or because of the poor expansion of the lung. Vocal fremitus rarely is diminished or absent. If the consolidation begin in the deeper parts of the lung—the so-called "central pneumonia"—the physical signs may be delayed for several days, making the diagnosis often extremely difficult.

In old people, in whom respiration is feeble, the only physical signs may be the subcrepitant râle and the feeble breathing. Dulness is frequently absent in the aged, in whom senile changes in the ribs allow of increased resonance on percussion.

3. *Stage of Resolution.*—The dulness becomes less and less marked, bronchial voice and breathing become broncho-vesicular and finally normal, vocal fremitus diminishes to the normal limit, and moist bronchial râles appear.

Exceptional Signs.—The dulness may change to tympany, which is often the earliest sign of resolution. Crepitant and subcrepitant râles, if formerly absent, may be heard, from increased lung-expansion. In some cases the moist bronchial râles are not present. Should thickening of the pleura persist, slight dulness, feeble breathing, and the crepitant râle may be present for a considerable time.

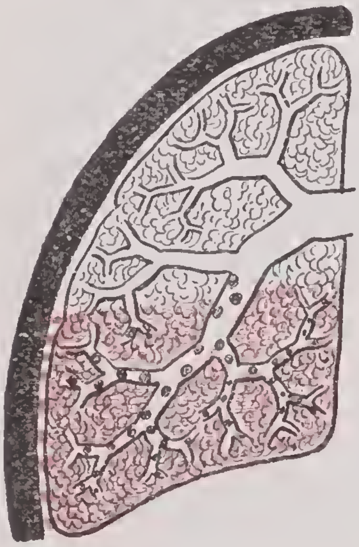


FIG. 33.—Physical signs of lobar pneumonia during the stage of resolution: dull tympany or tympany; broncho-vesicular voice and breathing, becoming harsh, feeble, or normal; vocal fremitus, becoming normal; crepitant and subcrepitant pleuritic râles; moist bronchial râles.

Complications.—*Pleurisy* with effusion is to be suspected should there be disproportional rapidity of breathing, and a continuance of fever beyond the natural duration of pneumonia. Empyema is marked by the occurrence of septic symptoms, erratic chills, irregular temperature curve, and sweating. The physical signs are those of fluid in the pleural cavity, but bronchial voice and breathing may be heard below the level of the fluid. In doubtful cases an exploratory aspiration should be resorted to.

Abscess of the lung occurs in about 1 per cent. of cases, from added infection by suppurative microbes. It is usually seen in debilitated subjects. Septic symptoms are present. There is an expectoration of pus, often fetid, containing shreds of lung-tissue, and prostration is extreme. In old people there may be no marked symptoms.

The physical signs are those of pulmonary cavities—tympanitic or "cracked-pot" note, cavernous voice and breathing, with moist and gurgling râles. These signs are often present over a consolidated lung-area containing a large bronchus with adherent pleural surfaces, so that a diagnosis by physical signs alone is often impossible. Many so-called cases of "abscess of the lung" complicating pneumonia are really acute phthisis with the rapid formation of cavities. In doubtful cases a bacterial examination of the sputum for the tubercle bacillus should be made.

Gangrene of the lung is somewhat less common than abscess. The expectoration is a greenish or brownish fluid, of fetid gangrenous odor, containing shreds of decomposed lung-tissue and crystals of fatty acids. Constitutional symptoms are usually pronounced.

Pericarditis in some cases adds typical symptoms—dyspnœa, rapid and feeble pulse, venous congestions, and characteristic physical signs. In other cases the diagnosis is less evident. Pericardial râles may be simulated by the rubbing together of overlying inflamed pleural surfaces at each systole of the heart, and signs of fluid may be obscured should the pneumonia involve the overlying portion of the lung.

Endocarditis may be either simple or malignant. The latter is to be suspected should septic and embolic symptoms be present. Pneumonia occurring in a patient the victim of chronic endocarditis may upset compensation and lead to heart failure. During the pneumonia pre-existing murmurs of valvular disease may be absent entirely.

Meningitis may complicate pneumonia with especial frequency at different times and in different places. It may run its course with or without typical symptoms. This complication may be mistaken for epidemic cerebro-spinal meningitis with complicating pneumonia, for tubercular meningitis with lesions in the lungs, and for uncomplicated pneumonia in children with marked cerebral symptoms.

Pulmonary œdema usually ushers in the fatal issue. The heart failing, the right ventricle becomes more and more distended, with consequent congestion and œdema of the lungs. The pulse becomes rapid and feeble; respirations are shallow, rapid, and attended by noisy bubbling sounds. The expectoration becomes profuse and frothy, and it may be blood-stained. Signs of deficient aëration of blood appear, and consciousness is lost some few hours before death.

Sudden heart failure may occur at any time, even after defervescence. The most usual time is about the time of crisis. In some cases thrombosis of the coronary or of the pulmonary artery may be found; in other cases there seems to be no assignable cause.

Chronic interstitial pneumonia, or fibroid induration, may in rare cases result from the organization of the inflammatory exudate into connective tissue, rendering the affected area of lung permanently consolidated.

Prognosis.—The average mortality in hospital cases ranges about 25 per cent.; it is somewhat less in private practice. Pneumonia of the apex, an extensive lesion, and old age render the outlook serious, and the complications increase the mortality to a considerable degree. Alcoholism must be considered in making the prognosis. According to the New York Hospital records, the mortality in non-alcoholic cases was 25 per cent.; in slightly alcoholic subjects, 33 per cent.; in those with a marked alcoholic history, 72 per cent. Patients subject to chronic Bright's disease are apt to die, especially if they be addicted to alcohol. At the New York Hospital 4 such patients recovered and 36 died. The earlier the patient is put to bed, the better is the prognosis.

Treatment.—Pneumonia is a self-limited disease and has no specific medication. In many cases no medicines at all are required. The patient should be kept in bed until at least five days after the temperature has become normal, and on a liquid or light diet. Restlessness and sleeplessness may be controlled by phenacetine, sulphonal, chloralamide, or by a Dover's powder given at night. Pain in the side may be relieved by hot poultices or by a hypodermic injection of morphine. Poultices need not be applied, however, as a routine measure; in fact, many patients are more relieved by cold applications to the chest by ice-bags—a treatment which appears to exert a beneficial effect upon the disease itself.

All attempts to abort the disease have proved futile. Large doses of calomel—from 25 to 40 grains placed dry on the tongue—seem to exert a beneficial effect upon the course of the disease in some cases, producing a sedative effect, but there is always the risk of producing salivation. Venesection at the outset in robust subjects with pronounced inflammatory symptoms is often of the greatest service, relieving the dyspnœa and the cerebral symptoms and reducing the fever. In the later stages, when the heart is beginning to fail, and cyanosis and symptoms of dilatation of the right ventricle appear, venesection may be employed as a last resort.

As an arterial sedative tincture of aconite or tincture of veratrum viride has seemed to be of service if given at the outset, but in robust patients venesection is preferable.

The temperature, being of relatively short duration, rarely requires much treatment. The use of internal antipyretics as a routine is to be deplored, on account of their depressant effect. High and prolonged temperature should, however, be controlled. For this purpose hydrotherapy is the best treatment to be employed—either cold sponging, the cold pack, cold applications to the chest, or even the bath at 70° F. By the use of the bath the temperature is reduced, the pulse becomes stronger, and cerebral symptoms are markedly relieved.

By far the most important treatment is that to sustain the action of the heart. Should the pulse become rapid and feeble and the second pulmonary sound weak, alcohol should be given freely, in doses sufficient to accomplish the end desired. In the aged and in alcoholic subjects it should be given from the start. The use of digitalis is frequently disappointing from its contractile power over the small arteries, unless it be combined with an arterial relaxant, such as nitroglycerin or iodide of potassium. A good combination is—

℞.	Potassii iodidi,	gr. v;
	Ext. digitalis fluid.,	♏j;
	Ext. convallariæ fluid.,	♏xx.—M.

Sig. Such a dose every three hours.

As a cardiac tonic strychnine is rapidly gaining in favor. It may be combined with digitalis and aconitine with advantage, as in the following prescription:

℞.	Strychnine sulphate,	gr. $\frac{1}{60}$;
	Digitaline,	gr. $\frac{1}{120}$;
	Aconitine,	gr. $\frac{1}{120}$.

Such a combination in pill form may be given every two or three hours until the pulse is reduced to about 100, but with such a form of treatment the patient should be watched carefully. Ammonia or camphor may also be employed.

Should heart failure with increasing difficulty of breathing occur, free stimulation, cupping of the chest, and the administration of pure oxygen gas should be employed ; in some cases free venesection proves satisfactory.

Complicating delirium tremens calls for free stimulation, for sedatives such as bromide, chloral, or hypodermic injections of morphine, and frequently for physical restraint. The sedative and tonic effect of the cold bath is often surprising in these cases.

Expectorants serve only to upset the stomach, although in some cases of tardy resolution pilocarpine may be employed, its depressant effect being carefully guarded by stimulants.

Experiments recently made by Drs. G. and F. Klemperer upon the toxines of pneumonia are of wonderful and vital interest. These observers found that rabbits could be rendered immune by hypodermic injections of heated pneumococcus cultures, and that the blood-serum from these rabbits, injected into patients suffering from pneumonia, produced an immediate curative effect upon the disease. More recently the blood-serum of patients who had just passed the time of crisis has been used for the injections, with equally brilliant results, the theory being that the serum from immune animals or from convalescent patients contains an antitoxine capable of neutralizing the toxine caused by the growth of the pneumococcus in the body. The latter, known as "pneumotoxine," has already been isolated, but the supposed antidotal toxine, the "antipneumotoxine," has not as yet been obtained in a chemically pure state.

BRONCHO-PNEUMONIA.

Eitology and Synonyms.—Broncho-pneumonia is the regular pneumonia of young children, but it may be seen in adults, and it is not uncommon in old people. Primary cases follow exposure to wet and cold or the inhalation of irritating chemical vapors; they are more common in the winter and spring months and among the debilitated tenement-house and asylum children. Secondary cases accompany many of the infectious diseases, especially measles, scarlet fever, whooping-cough, and diphtheria. Broncho-pneumonia may occur in any disease which keeps the patient in bed, the dorsal decubitus preventing free expectoration, and the foul mouth of fevers allowing the growth of bacteria in the mouth. The bacteria enter the lung with the inspired air and infect it. Careful cleansing of the mouth in prolonged fevers will often prevent the occurrence of this so-called "aspiration-" or "inhalation-pneumonia." "Deglutition-pneumonia" is that form of pneumonia produced by the passage of food or drink into the bronchi from choking at table, in deep coma, or from tracheotomy, intubation, or cancer of the larynx or the œsophagus. Suppuration or even gangrene may result in such cases. Patients with advanced emphysema are liable to subacute attacks of broncho-pneumonia. Tubercular broncho-pneumonia will be considered under a separate heading. Broncho-pneumonia may be produced either by the ordinary pneumococcus or by the streptococcus of suppuration. *Synonyms:* Lobular pneumonia; Catarrhal pneumonia; Capillary bronchitis.

Pathology.—The lesion consists of a bronchitis and a pneumonia. The *bronchitis* is general, involving the bronchi of both lungs. The large, the medium, and the smallest bronchi are all involved, although not always to the same extent. The mucous membrane of the bronchi show ordinary catarrhal inflammation; the walls of the smallest bronchi are thickened by an infiltration of new cells, and may be dilated; the smaller bronchi may contain pus. The *pneumonia* differs from lobar pneumonia in the location of the affected areas and in the regular involvement of the connective-tissue walls of the air-cells. Around the bronchi are zones of consolidated lung-tissue, varying in size from a pin's head to that of a pea. These areas of peri-bronchitic hepatization may be so slight as almost to escape notice, so that the lesion appears to be bronchitis alone. These cases were formerly described as "capillary bronchitis." In other cases the areas are larger, so that the lungs are filled by these isolated nodular bodies; while in yet other cases, the "pseudo-lobar form," the areas are large and confluent, so that the greater part of the lobe is rendered solid. The cut surface of a broncho-pneumonic patch is smooth, lacking the granular appearance of the hepatization of lobar pneumonia. Gray hepatization is but rarely seen.

Microscopic examination shows the air-spaces filled with fibrin, pus, epithelial cells, and red blood-cells.

In children there is a relatively greater proportion of epithelial cells in the exudate than in adults. The walls of the air-spaces are thickened and are infiltrated by small round cells or by fibrin, pus, and epithelium.

The lung-tissue between the nodules may be either normal or congested and œdematous, or it may be the seat of a diffuse pneumonia in which the air-spaces are partially filled with fibrin, pus, epithelial cells, and red blood-cells, or the air-spaces may be collapsed. These areas of collapse or atelectasis are depressed below the surrounding tissues and are of a bluish or blue-brown color. These areas may be small and may only surround the peribronchitic nodules, or the greater part of a lobe may be involved. In recent cases the air-spaces may be inflated by a blow-pipe inserted into the bronchus leading to them.

The pleura covering the peripheral pneumonic patches is covered with fibrin. The bronchial glands are almost always swollen and inflamed. This condition may persist, rendering the glands susceptible to an added tubercular infection, from which systemic infection may result. Many cases of tuberculosis in asylums follow epidemics of measles, the sequence being measles, broncho-pneumonia, inflammation, tubercular infection of the inflamed bronchial glands, general miliary tuberculosis.

There is a common misapprehension that the inflammation extends from the bronchi into the air-spaces which open into them. In point of fact, it is the air-spaces surrounding the axis of the bronchi that are involved. If hot knitting-needles be run into a loaf of bread, the path of each needle will represent a bronchus, while the charred bread surrounding its track will represent the position of the peribronchitic pneumonia.

The interstitial inflammation of the walls of the bronchi and the air-spaces is a special feature of broncho-pneumonia. It is of especial importance from its tendency to become chronic, by the changing of the infiltrating cells into developed connective tissue.

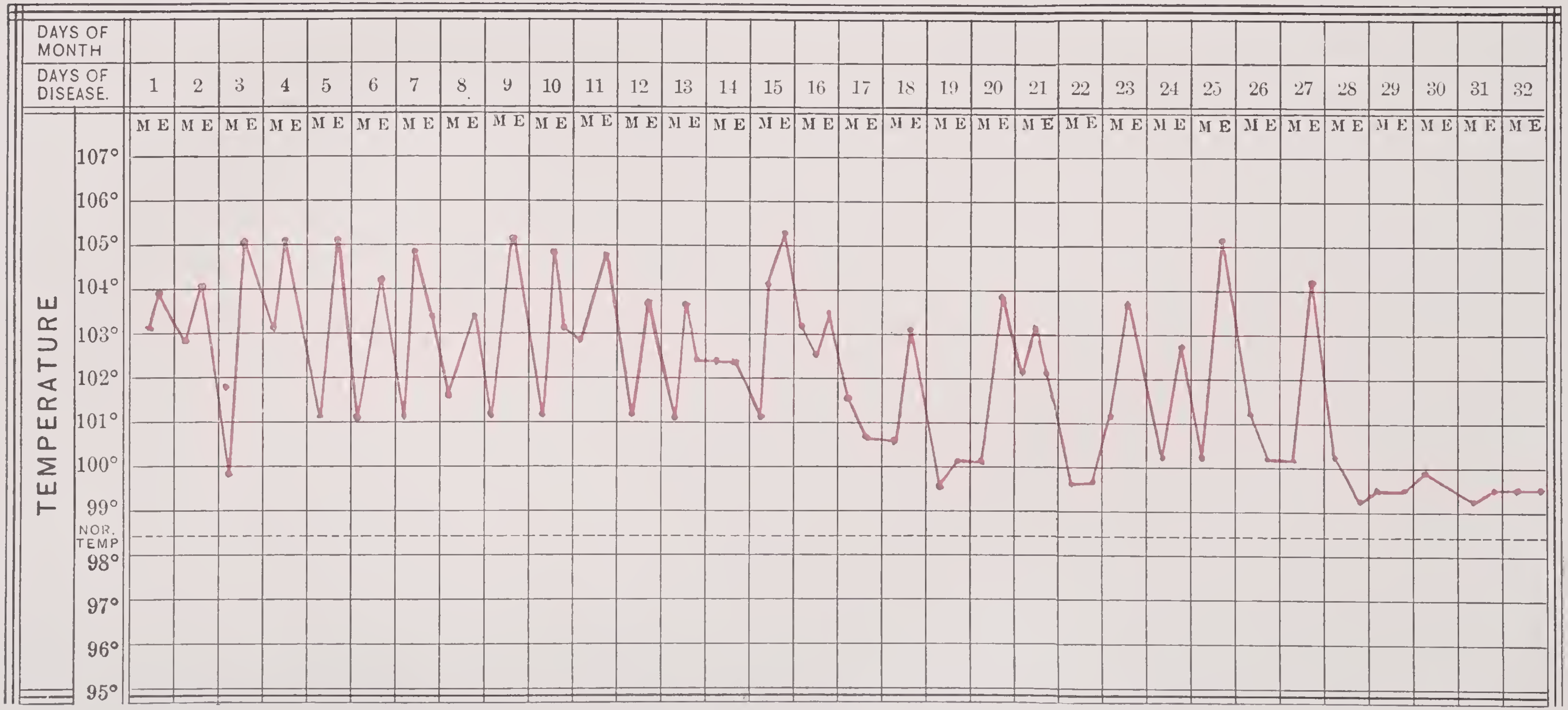
Symptoms.—*In young babies* fever, prostration, and rapid breathing are the only constant symptoms. There may be no cough, and physical signs are not present. The disease runs almost regularly a fatal course within a few days.

Mild cases in children may resemble severe ordinary bronchitis. In broncho-pneumonia the general symptoms, however, are more severe, and the physical signs point to an involvement of smaller bronchi than in bronchitis alone. A temperature of 102.5° or 103° F. persisting for three days gives evidence of more than a simple bronchitis.

Severer forms in children may begin gradually or abruptly —gradually by the extension of a simple bronchitis, or abruptly by convulsions or vomiting. Should the pneu-

PLATE 20.

BRONCHO-PNEUMONIA.



Broncho-pneumonia: prolonged course; recovery.

monia be secondary to some well-marked infectious disease, the onset is somewhat obscured.

When the disease is established there is a fever which is usually remittent in character, often markedly so. There is no typical curve, as in lobar pneumonia, and there is no day of crisis. The height of the fever is often proportional to the severity of the attack, but some patients do badly whose temperature ranges between 99° and 100° F. The circumstances under which these low temperatures are met with are early infancy, low vitality, and great prostration with little reactionary power. The pulse becomes rapid, frequently ranging from 140 to 180 in many children. Respirations may be as high as 50 to 80 to the minute, and they are marked by inspiratory dilatation of the alæ nasi. There is often an expiratory moan or grunt. There may be audible bronchial râles. The dyspnœa may make it difficult for the child to nurse. There is frequently cough, which may be painful. The sputum is that of bronchitis; it is usually swallowed, but it may be vomited up. Rusty sputum is not present. In older children the sputa may be blood-streaked. The face is flushed, the skin is dry, the tongue is coated; vomiting is frequent, and it may interfere with the proper feeding of the child. There may be restlessness, sleeplessness, or mild delirium, depending on the fever and the disposition of the child. The urine may contain small amounts of albumin and casts. In cases developing during

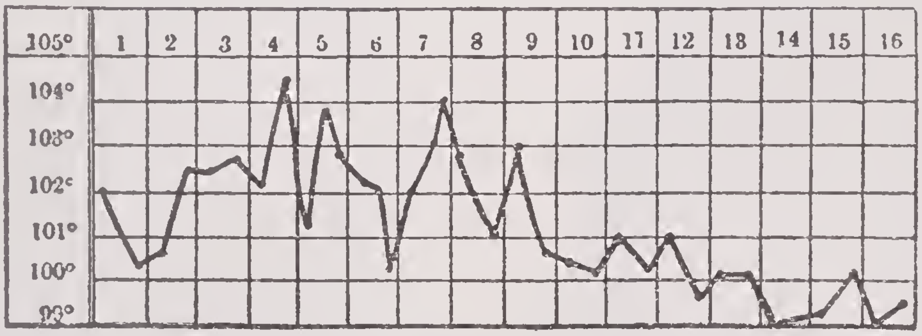


FIG. 34.—Typical broncho-pneumonia of the milder form (Holt).

some severe disease, constitutional symptoms may be obscured, the rapid breathing alone attracting attention to the lung.

Cerebral Cases.—There are cases in which the cerebral symptoms may be so pronounced as to obscure those of pulmonary origin. There may be headache, repeated convulsions, delirium, photophobia, retraction of the head, and muscular twitchings. Pulmonary symptoms appear later in the disease in the majority of cases, with a subsidence of the cerebral symptoms. The diagnosis from meningitis in many cases is difficult. The points in favor of pneumonia are absence of tuberculosis or of suppurative ear disease, a higher respiration-rate, absence of paralysis, a more rapid pulse, and the presence of physical signs. The cerebral symptoms, moreover, are neither so severe nor so continuous as in meningitis, and they disappear with defervescence.

Physical Signs.—In all cases signs of bronchitis are present—coarse and subcrepitant râles, especially at the bases, with sibilant and sonorous breathing. If the pneumonic patches be small and scattered, there may be no added signs, so that the diagnosis from bronchitis will be made by other means. If the pneumonic patches be larger and nearer one another, there will be a tympanitic or dull note with harsh breathing. If the patches so coalesce as to involve a larger area, there will be dulness, bronchial voice and breathing, and increased vocal fremitus. Fibrin on the pleura gives rise to crepitant and subcrepitant râles of superficial quality.

Course and Termination.—The disease may terminate—(1) *In resolution.* The disease continues for from two to three weeks in the majority of cases, the temperature falling by lysis. In some cases the symptoms continue for from six to eight weeks before recovery. Resolution is slower than in lobar pneumonia, being rarely completed in less than from seven to fourteen days.

(2) *In death from asphyxia.* In cases that are doing poorly the dyspnœa becomes more distressing, the skin is bluish, and the pulse becomes more and more rapid and feeble. As the child gradually succumbs to carbon-dioxide poisoning the dyspnœa becomes less apparent, distress gives way to stupor alternating with restlessness, the cyanosis deepens, the bronchi fill with mucus, and death results from heart paralysis.

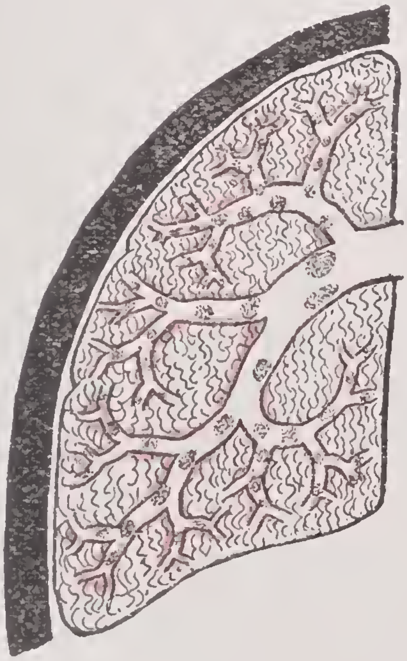


FIG. 35.—Broncho-pneumonia: zones of peribronchial consolidation very slight; physical signs of bronchitis ("capillary bronchitis").

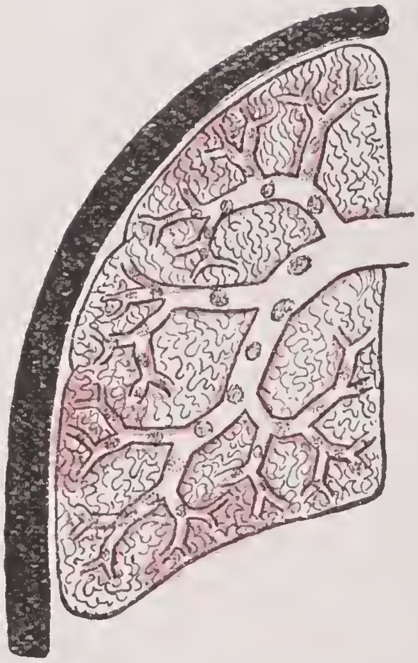


FIG. 36.—Broncho-pneumonia: zones of peribronchial consolidation more extensive; physical signs of general bronchitis and of small localized areas of consolidation.

(3) *In suppuration or gangrene.* Either termination is rare except in the aspiration and deglutition forms.

(4) *In chronic interstitial pneumonia.* In these cases the cells infiltrating the walls of the bronchi and of the air-cells become organized into connective tissue. In this way the walls of the bronchi are thickened and dilated and are surrounded by zones of connective tissue that represent the

former areas of peribronchitic pneumonia. By the coalescence of these zones more or less of the lung is rendered permanently unfit for use. The pulmonary pleura is usually much thickened. Such a lung is exceedingly apt to develop tubercular changes in the course of time.

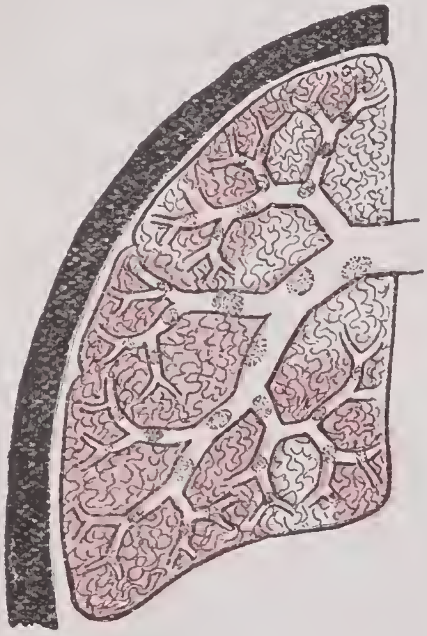


FIG. 37.—Broncho-pneumonia: peribronchial consolidation extensive, giving the physical signs of consolidation of large areas, resembling lobar pneumonia.

In some cases the pneumonia runs an acute course, and in one or two weeks the temperature falls and the child becomes better but cannot be said to be actually convalescent. There continues to be a moderate irregular fever; the cough continues and emaciation becomes more marked.

After a few months some of these cases recover, though with small areas of interstitial pneumonia which may give no further trouble or which may cause repeated attacks of bronchitis, while in other children tuberculosis develops, either from infection from tubercular bronchial glands or from a local infection of the disabled lungs by the tubercle bacillus.

In other cases the fever, cough, and emaciation continue, hectic develops, and the child dies emaciated and exhausted. The course of the disease is that of chronic pulmonary phthisis, but at the autopsy there is found extensive interstitial pneumonia with large bronchiectatic cavities.

BRONCHO-PNEUMONIA IN ADULTS.

The disease in adults presents a variety of clinical forms which are thus described by Delafield:

1. There is first ordinary bronchitis for several days, but,

instead of recovering, the patient continues to cough and to feel sick, and at some part of the chest there is found a small area of dulness and high-pitched voice. The consolidation lasts but a short time, and the patient regularly recovers.

2. The patient is seized by a chill; there are rapid and high fever, pains in the back and the chest, great prostration, rapid and feeble pulse, rapid and insufficient breathing, cough with mucous and blood-stained sputa, sleeplessness, restlessness, and delirium. The urine contains albumin and casts; the skin is cyanosed; the viscera are congested. Over both chests the percussion-note is normal, exaggerated, or dull. Coarse subcrepitant and crepitant râles with sibilant and sonorous breathing are heard. The disease lasts for one or two weeks and is apt to prove fatal.

3. A form of broncho-pneumonia resembles lobar pneumonia. There is a general bronchitis, with broncho-pneumonia and consolidation of one or more lobes. Compared with lobar pneumonia, the invasion is more gradual, the pulse is more rapid, cerebral symptoms are more constant, the sputum is that of bronchitis, the physical signs are delayed in their appearance, the duration is longer, and resolution is slower.

4. A form resembles tubercular broncho-pneumonia. The invasion is gradual and the disease is protracted for weeks. The patients have fever with evening exacerbations and night-sweats, cough with muco-purulent expectoration which does not contain tubercle bacilli, and there is a loss of flesh and of strength. Physical signs show bronchitis with localized areas of consolidation. Some patients recover after a number of weeks; in others the disease proves fatal.

5. Patients with emphysema may develop a subacute broncho-pneumonia which is often fatal.

6. Broncho-pneumonia, especially of the lower lobes, may be seen in infectious diseases, injuries, and operations which cause congestion of the lungs and allow of the inhalation of streptococci.

The **prognosis** depends—(1) Upon the age. The disease is generally fatal in young infants. The mortality is 50 per cent. under one year, 40 per cent. between the first and the third year, and 25 per cent. between the third and the fifth year. (2) Upon the severity of the attack. Mild cases may be no more fatal than bronchitis. (3) Upon the general nutrition of the child, being worse in asylum- and tenement-house cases. (4) Upon the nature of the primary disease, being worse with whooping-cough, measles, or diphtheria. Aspiration- and deglutition-pneumoniasare usually severe. An absolute prognosis should never be made, as the sickest child may recover and apparently mild cases may do badly.

The prognosis in cases of chronic broncho-pneumonia depends upon the amount of lung disabled, the possibility of avoiding tubercular infection, and the care that can be taken of the case.

Treatment.—Much can be done to prevent the disease. Convalescent fever patients, and especially those convalescing after measles or whooping-cough, should be guarded carefully from catching cold. All bronchial affections in young children and in the debilitated and the aged should be attended to properly, especially in the winter and spring months. The mouth and the teeth of all fever and bedridden patients should be kept scrupulously clean by frequent washings with antiseptic solutions. Hypostatic congestion is to be prevented by occasional change of position in bed.

In general terms, the treatment of broncho-pneumonia is that of lobar pneumonia, except that the patient is usually a young child and that the bronchitis is an additional feature. There is, moreover, less danger of heart failure than in lobar pneumonia. The patient should be put to bed in a room having a temperature of about 68° or 72° F., the air being kept moist by a steam-kettle or an atomizer. The diet should be fluid during the febrile stage, and the patient should be allowed cool water sufficient to quench the thirst. At the outset the bowels should be opened, calomel (gr. $\frac{1}{10}$ every hour until an movement occurs) being generally advisable. For the febrile symptoms an ordinary saline fever-mixture may be employed, such as citrate of potash, liquor ammoniæ acetatis, or spiritus ætheris nitrosi. If there be high fever with bounding pulse, minim doses of tincture of aconite may be employed every 1 to 3 hours. Internal antipyretics are not to be recommended. Should the fever need reduction, it is best to employ the cold bath or the wet pack, beginning with a tepid temperature and gradually reducing it to 80° or 75° F.

For the bronchitis counter-irritation may be applied to the chest by turpentine and sweet oil, by mustard poultices made thin and light, or by chloroform liniment. The chest may be covered with the oil-silk jacket, which consists of a flannel shirt over which are sewn strips of oil silk. Poultices are heavy and are not so much employed now as formerly. Expectorants are to be given in the majority of cases, as in simple bronchitis, but disturbance of the stomach and continual nausea should be avoided. If the child has trouble in bringing up the mucus, an occasional emetic is often valuable, provided the heart be acting well.

In commencing asphyxia and heart failure inhalations of pure oxygen gas are to be recommended, and vigorous heart-stimulation is to be employed, digitalis, strychnine, and small doses of brandy being used for this purpose. Under these circumstances a hot mustard bath for the entire body is often beneficial.

Pain and distressing cough frequently require small doses of Dover's powder or codeine, but opium must be given with extreme caution to young children.

Cerebral symptoms are best treated by phenacetine, sodium bromide, or chloral, and by the cold pack. If resolution be delayed or if the danger of a persistent interstitial pneumonia threaten, a vigorous tonic treatment of iron, quinine, and cod-liver oil is to be employed, with strict attention to the diet and the assimilation of food. Should these means prove inoperative, a change of air is advisable.

INTERSTITIAL PNEUMONIA.

Definition.—Interstitial pneumonia is a chronic inflammation of the connective-tissue framework of the lung, resulting in the production of new connective tissue and the obliteration of the air-spaces.

Etiology.—The disease is part of the lesion of chronic pulmonary phthisis, of syphilitic pneumonia, and of substantive emphysema. It occurs as the result of chronic bronchitis, pleuritic adhesions, broncho-pneumonia, lobar pneumonia, and the continual inhalation of solid particles in the air in various occupations. To the disease caused by inhalation the term "pneumonokoniosis" has been applied, while special names are given according to the nature of the inhaled particles: *Anthracosis*, due to the inhalation of coal-dust; *siderosis*, due to iron-dust; *chalicosis*, due to stone-dust, etc. The names of various occupations have also been attached, such as "miners' phthisis," "millers' lung," "masons' lung," "stone-cutters' phthisis," "grinders' rot," etc.

Pathology.—The lesion consists in the growth of connective tissue which replaces the normal lung structure. The walls of the air-spaces and of the bronchi are usually first involved by connective-tissue thickening, so that the air-spaces are diminished in size, are frequently deformed by polypoid projections into them of connective tissue, and may be effaced entirely. The walls of the bronchi, losing their elasticity, may dilate to form bronchiectatic cavities whose lining may be mucous membrane, connective tissue, or a suppurating surface. Similar connective-tissue changes are usually seen in the pleura, which becomes thickened and adherent.

The new connective tissue, having scanty blood-vessels, replaces vascular lung-tissue, so that, the number of blood-channels between the right and the left heart becoming reduced, there may either be a compensatory hypertrophy of the right ventricle, or its dilatation with general venous congestions. The unaffected portions of the lung are apt to be emphysematous.

The appearance of the lung varies with the cause of the pneumonia.

1. *Interstitial Pneumonia due to the Inhalation of Dust.*—The connective tissue thickening occurs in the walls of the bronchi and of the surrounding air-spaces. Later there may be areas of different interstitial pneumonia, rendering more or less of the lung solid. The affected areas are stained by the deposit of the irritating dust-particles; the bronchial glands are inflamed and contain the offending particles. Bronchiectatic cavities frequently occur.

2. *Interstitial Pneumonia following Broncho-pneumonia.*—Here the chronic lesion follows the localities of the acute form. The walls of the bronchi are involved and frequently are dilated; there are peribronchial fibrous nodules, with occasionally diffused areas of interstitial pneumonia. The process may be entirely peribronchial or it may involve a whole lobe.

3. *Interstitial Pneumonia due to Lobar Pneumonia.*—These are the rare cases in which the exuded products in the air-

spaces become organized, so that the lobe is rendered permanently solid.

4. *Interstitial Pneumonia following Chronic Pleurisy.*—In this form the pleura is much thickened, and from it bands of connective tissue pass into the substance of the lung.

5. *Interstitial Pneumonia following Chronic Bronchitis.*—Here the walls of the bronchi are thickened and dilated with fibrous nodules about the bronchi.

The **symptoms** begin gradually. There is a chronic bronchitis with a cough and with mucous or muco-purulent expectoration which may be fetid. There may be small repeated hæmoptyses. If there be bronchiectatic cavities, the expectoration will be more profuse, and from time to time, frequently after bending over or after any change in position, a large quantity of secretion will be expectorated.

Fever is not present except during acute exacerbations of the bronchitis and from suppurating bronchiectatic cavities. In the latter case the fever may assume the hectic type, with night-sweats and emaciation similar to the septic symptoms so commonly observed in pulmonary phthisis.

Pain in the chest is common if the pleura be involved. Dyspnœa is usually present, depending upon the extent of lung involved and the condition of the right heart. Should dilatation of the right heart occur, there will be the general venous congestions regularly observed in this condition.

The **physical signs** vary according to the actual condition of the lung. The bronchitis gives rise to coarse and subcrepitant râles with sibilant or sonorous breathing.

The fibrous areas give the regular signs of consolidation—dulness on percussion, increased vocal fremitus, and bronchial voice and breathing. If the areas be small, the note may be tympanitic and the breathing sounds may be feeble or harsh or broncho-vesicular.

Dilatation of the bronchi gives rise to the physical signs of cavities—tympanitic or "cracked-pot" note, more rarely a note of amphoric quality, with cavernous breathing, intensified voice, and bubbling râles of large size.

The thickened pleura causes dulness or flatness on percussion, while vocal fremitus, breathing, and voice are diminished or lost. There are also creaking, rubbing friction-sounds of all kinds.

From shrinkage of the connective tissue the affected areas are reduced in size, so that there is a retraction of the chest-wall and limited respiratory expansion, in strong contrast to the unaffected side. There may be overlapping of the ribs and spinal curvatures. The heart may be displaced by being drawn to the affected side, or the area of impulse may be abnormally visible should the lung over it be retracted.

Prognosis.—The disease lasts for years, the more severe cases being semi-invalids. There is always danger of an added tubercular infection, to which these lungs are exceedingly liable. If large areas of lung are involved, there is always danger of dilatation and failure of the power of the right heart. Patients, moreover, are apt to do badly if they develop acute inflammation of the remaining portions of the lung.

Treatment.—The only thing that can be done is to send the patient to a mild climate where the bronchitis will not be aggravated by exposure to cold and dampness. If this cannot be done, the general nutrition of the patient should be improved in every way, and the chronic bronchitis treated.

INTERLOBULAR EMPHYSEMA.

Etiology.—Interlobular emphysema usually follows severe expiratory efforts, as in whooping-cough, and is most frequently seen in the broncho-pneumonia of young children.

The disease has also occurred as the result of straining efforts with closure of the glottis in parturition, excessive muscular exertions, and convulsions.

Pathology.—By rupture of the air-spaces air escapes into the interlobular septa, rupturing into the pleural cavity to cause pneumothorax, or extensively infiltrating the connective-tissue septa and compressing the parenchyma of the lung. In rarer cases it may make its way into the mediastinum and extend up the course of the trachea to the subcutaneous tissues of the neck.

The **symptoms** are not distinctive. Pneumothorax may result, and severe cases may be followed by sudden death.

VESICULAR EMPHYSEMA.

There are three distinct forms of vesicular emphysema—compensatory, substantive, and senile.

Compensatory Emphysema.—When part of the lung is so disabled that it cannot expand fully, the remaining portions have to expand or the chest-wall will fall in. Compensatory emphysema consists, then, simply in an over-stretched condition of the lung, with distended air-vesicles whose walls are thinned. It occurs as a temporary condition in pneumonia, broncho-pneumonia, and with pleuritic effusions. When the cause is more permanent, as with phthisis, pleural adhesions, and interstitial pneumonia, groups of air-vesicles may remain permanently distended. This form is a normal compensatory process, gives no symptoms, subsides with the subsidence of the original disease, and is of no practical detriment to the patient.

Substantive Emphysema.—**Etiology.**—Much uncertainty exists as to the true nature of substantive emphysema. Formerly it was regarded as a mechanical over-inflation of the lung from forced inspiratory or expiratory efforts. This condition argued some weakness of the lung-tissue, so that it was supposed that, to account for the over-inflation, there must be a congenitally weak lung-tissue; this theory appeared to be borne out by the markedly hereditary character of the disease. It was found, however, that there were cases of emphysema without any dilatation of the air-spaces, so that this could not be regarded as an essential feature. Delafield describes the disease as a chronic interstitial inflammation of the lung, with which condition more or less dilatation of the air-spaces is usually, but not invariably, associated. His studies have been so extensive and thorough that his descriptions of the disease process will be followed to the exclusion of other theories.

Pathology.—The lungs are large, downy or feathery to the feel, and do not collapse when the chest is opened. Enlarged air-vesicles are usually visible, especially along the anterior margins, and on the inner surface of the lobe near the root of the lung. In some cases, however, there is no dilatation of the air-spaces, and these cases may even be attended by most marked symptoms of emphysema during life. The walls of the air-spaces are thickened in some parts of the lungs, thinned in others, and the epithelial cells lining them are often increased in size and number. Atrophic changes in the walls of the vesicles allow of perforation, so that a number of vesicles may merge into a common air-chamber. With the atrophy of areas of the vesicular wall there is a disappearance of the capillaries coursing over it, so that the number of channels between the right and the left heart becomes materially reduced. In many cases there exists a contraction of the smaller arteries, which still further increases the pulmonary obstruction. The septa between the lobules, the connective tissue around the bronchi and the blood-vessels, and the pulmonary pleura are often considerably thickened, and frequently there are adhesions between the pleural surfaces. There is regularly a chronic catarrhal bronchitis, but bronchiectasis is not common. There may be endarteritis of the branches of the pulmonary or bronchial arteries, and it is supposed that in many cases spasmodic contraction of these arteries occurs during the life of the patient.

Secondary lesions result from the mechanical obstruction to the pulmonary circulation, both from the disappearance of some of the capillaries, and also from the contraction of the small arteries; these lesions consist in the hypertrophy and dilatation of the right side of the heart. If dilatation and heart failure occur, there will be venous congestions of the skin, the serous membranes, and the viscera. The condition of the right heart—whether compensatory hypertrophy occurs and remains established—is of the greatest importance to the patient.

Associated lesions are usually found. They consist of chronic endocarditis, chronic endarteritis, and chronic diffuse nephritis. These lesions depend, as does emphysema, upon the slow production of connective tissue replacing pre-existing tissues, and they are therefore apt to be associated in the same patient.

Symptoms.—Of the regular symptoms, dyspnœa is the most prominent. At first the dyspnœa is induced only by exertion, by indigestion, or by attacks of bronchitis; later it becomes more steady and troublesome. Many of the attacks of dyspnœa appear to be due to contraction of the small arteries of the lung.

Symptoms of bronchitis are present, especially in the winter months. The patient has a paroxysmal, ineffectual cough, with muco-purulent expectoration. Exacerbations of the bronchitis are accompanied by an increase of the cough and the expectoration, and possibly by slight fever, night-sweats, and rarely by small hæmoptyses. Should bronchiectatic cavities form, they are marked by a characteristically profuse expectoration after change of position and by physical signs.

Symptoms of asthma are commonly present; they are usually most severe during the exacerbations of the bronchitis.

The respirations are labored and wheezy; expiration is unduly prolonged. The obstruction in the pulmonary system allows of cyanosis, often of an extreme grade, but it is not incompatible with comparative comfort.

When compensation fails and the right heart dilates, general venous congestions are gradually developed—congestion and œdema of the skin, congestion of the stomach, the liver, and the kidneys, and general dropsy.

The symptoms of chronic endarteritis, of chronic endocarditis, or of chronic diffuse nephritis may complicate the course of the disease and may even obscure the diagnosis.

Variations in the Course of the Disease.—The following clinical types of the disease are described by Delafield:

"1. Some patients for years have a winter cough, with expectoration of mucus and sometimes of a little blood. They are always a little short of breath when they exert themselves. After a time they have attacks of spasmodic asthma. Then the dyspnœa on exertion becomes more constant and more decided; the patients lose flesh and strength; venous congestion is established, dropsy, and death.

"2. Other patients are fairly well except when they have attacks of acute bronchitis. Such attacks may be mild, lasting a few days or a few weeks, with cough, mucous expectoration, sometimes hæmoptyses, asthmatic breathing, and a febrile movement; or the attacks may be severe, and last two or three months, and, in addition to the symptoms just mentioned, they develop venous congestion, albuminuria, and dropsy.

"3. In some patients there is a history of attacks of spasmodic asthma for a number of years before the symptoms of emphysema make their appearance.

"4. In some patients the evidences of emphysema are very slight for a long time. Then rather suddenly constant dyspnœa and venous congestion are developed, and the patients die in a few months."

Physical Signs.—Inspection may reveal nothing abnormal. In advanced cases there is an increase in the antero-posterior dimensions of the thorax, approaching the "barrel-shaped chest" in some cases. The chest rises and falls *en masse* and with evident muscular action, dyspnœa being evident. Lateral expansion is not well marked. The head inclines forward, the shoulders are rounded, the sterno-cleido-mastoid muscles are prominent, and the respiratory action of the diaphragm is increased. The thorax in emphysema has aptly been described as one of "permanent inspiration."

Pulmonary resonance may be unchanged or vesiculo-tympanitic, or there may exist an exaggerated resonance of a tympanitic quality, or the resonance may be of a variety of tympany of a dull quality. This latter note, often called "wooden," is highly characteristic. The percussion boundaries of the lung are increased in all dimensions, and, owing to the increased thickness of the covering lung, the borders of the heart are determined with great difficulty.

The characteristic breathing is feeble compared with the evident expansion of the chest; the expiration is much longer than inspiration, and is of a lower pitch. More rarely both inspiration and expiration may be harsh, loud, and high-pitched.

Sibilant and sonorous râles are usually present, and the bronchitis adds coarse mucous râles.

Prognosis.—Substantive emphysema is essentially chronic, its course extending over years. Moderate degrees of emphysysema are not incompatible with prolonged and active life, but in each case the questions must be, How much extra work is thrown upon the right heart? and, How long can compensation be maintained? Due regard should also be had for the presence of associated diseases—diseased arteries, heart, and kidneys.

Treatment.—Much can be done to check the course of the disease by sending the patient to some warm inland place for the winter months at least, where he can lead a life out of doors and not contract recurring attacks of bronchitis. The general health should be built up in every way; over-use of tobacco and of stimulants is to be interdicted; starches and sugars should be avoided.

Attacks of dyspnœa are best controlled by drugs that dilate the small arteries—chloral hydrate, iodide of potassium, and nitroglycerin. A good combination is as follows:

℞. Liq. morph. sulph. (Magendie),	♏j;
Tinct. belladonnæ,	♏iij;
Potassii iodidi,	gr. vij;
Spiritus ætheris comp.,	♏xv;
Aquæ,	℥ss.—M.

Sig. Such a dose every three or four hours.

Bronchitis is to be treated on general principles. Should the cough be harassing, the fluid extract of chekan, in dram doses every three hours, is often of the greatest service. The fluid extract, desiccated and given in capsule, is a convenient and pleasant form of administration. Terpin and the turpentine derivatives are of service should the expectoration be profuse.

When the right heart begins to fail and venous congestions appear, cardiac stimulants are indicated. Of these strychnine is especially useful. The bowels should be kept freely open, and all tendency to flatulent dyspepsia is to be combated by diet and drugs. Should the venous congestions be urgent and the patient be fairly robust, free bleeding is often followed by marked improvement.

GANGRENE OF THE LUNG.

Etiology.—Gangrene of the lung is caused by infection by means of the putrefactive bacteria. As these germs are so common in inspired air, a condition of impaired lung-vitality must be presupposed. The disease is most commonly caused by the entrance of organic foreign bodies into the bronchi, from food or other bodies being inhaled into the trachea, from "aspiration-" or "deglutition-pneumonia," or from the perforation of the lung by cancer of the œsophagus or of the stomach. It may follow cavities, bronchiectasis, or fetid bronchitis. It occasionally follows embolism or pressure of the branches of the pulmonary or the bronchial arteries, and it is one of the rare sequelæ of lobar pneumonia. Exceptionally it occurs in debilitated subjects, especially those with diabetes, without antecedent pulmonary disease.

Pathology.—A circumscribed and a diffuse form are recognized.

The *circumscribed* form occurs in single or multiple foci, usually in the lower lobe near the periphery. The gangrenous area is converted to a greenish-brown offensive mass surrounded by congested or consolidated lung-tissue. The neighboring veins are frequently filled with infective thrombi that may become detached, causing secondary foci in other parts of the body. In this way abscess of the brain may be developed. If the pleura be involved, pleurisy with a sanious or purulent effusion will result, or perforation of the pleura will lead to pyo-pneumothorax. Severe and even fatal hemorrhage will result from erosion of a large arterial branch. An intense general bronchitis always complicates the disease. Liquefaction of the gangrenous area rapidly occurs, and, the softened portions being coughed up, a cavity with ragged necrotic walls is left. Should the patient recover, a connective-tissue capsule forms, enclosing the cavity, which may subsequently contract.

The *diffuse* form may be so from the start or may follow the circumscribed form. A lobe or the greater part of a lobe, or even the entire lung, may thus become gangrenous. The diffuse form rarely follows lobar pneumonia.

Symptoms.—*Pulmonary.*—There is a cough with an expectoration which is fetid and usually abundant. On standing, the expectoration separates into three layers—an upper layer, frothy and opaque; a middle layer, clear and watery, and usually of a greenish or brownish tinge; and a lower layer, of a greenish sediment which consists of mucus, pus, shreds of elastic lung-tissue, granular matter, crystals of fatty acids, and bacteria.

Blood is often present in the sputa, and large hemorrhages may occur. Marked and characteristic fetor of the breath is rarely absent. Pain occurs if there be pleurisy.

Septic.—There is developed an irregular fever with prostration. The patient loses flesh and strength and passes into a typhoid condition. In cases in which the gangrenous area is encapsulated the septic symptoms are not so severe as one would expect.

The **physical signs** are those of consolidation over a certain area, followed by the signs of a pulmonary cavity. There are abundant moist bronchial râles. Physical signs of pleurisy with effusion or of pyo-pneumothorax may be present.

The **diagnosis** from fetid bronchitis is made by finding shreds of pulmonary tissue and elastic fibres in the sputum.

The **prognosis** is bad, but not hopeless. The course may be acute, or the disease may last for months.

Treatment consists in supporting the strength of the patient and in disinfecting the lung as thoroughly as our limited means will permit. The patient should wear continuously a Robinson inhaler saturated with equal parts of alcohol, creosote, and chloroform. Terpin and the derivatives of turpentine are often of great service, while myrtol, (gr. iiss in capsule every two hours) is warmly advocated.

If the gangrenous area be localized near the surface of the lung, injections of antiseptics may be made directly into it, and if the patient be in fair condition, the cavity may be opened through the chest-wall and drained.

ABSCESS OF THE LUNG.

Etiology.—Aside from the cases of purulent infiltration complicating lobar pneumonia or broncho-pneumonia, abscess of the lung may occur from the following causes:

1. From the introduction of foreign septic substances into the bronchi, as in aspiration- or deglutition-pneumonia, from septic diseases of the throat or the neck, from perforation into the lung of cancer of the œsophagus or the stomach or abscess of the liver, or from penetrating wounds of the thorax.

2. It may complicate lobar pneumonia or phthisis, or it may be due to the suppuration of an echinococcus cyst.

3. More commonly abscess of the lung results from infective emboli, the so-called "embolic" or "metastatic abscess." Multiple abscesses frequently occur with pyæmia or with malignant endocarditis involving the right heart. At first the lesion resembles an ordinary hemorrhagic infarction, but the embolic area rapidly becomes purulent and softens to form a cavity, while the pleura over it becomes infected, resulting usually in empyema or pyo-pneumothorax.

The **symptoms** are frequently masked by those of the original disease. Respirations, however, are quickened, the temperature becomes higher, and the patient is evidently much worse. Pain, if present, is due to the pleurisy. Empyema or pyo-pneumothorax may add its symptoms. The sputum is abundant, purulent, and contains shreds of lung-tissue. The odor is offensive, but not so markedly so as in fetid bronchitis or in gangrene of the lung. The constitutional symptoms are those of sepsis.

The **physical signs** at first are those of consolidation, becoming changed later to those of a cavity. Complicating pleurisy with effusion or pyo-pneumothorax adds its characteristic physical signs.

The **prognosis** is almost always fatal in embolic cases, but recovery occasionally occurs after pneumonia. Prompt and efficient treatment, however, may moderate the mortality rate.

The **treatment** should be entirely surgical, consisting in the opening and draining of the abscess-cavity. Recovery or improvement results in about one-half the cases so treated.

SYPHILIS OF THE LUNG.

Syphilis of the lung is a rare disease, but its actual occurrence is undoubted.

Pathology.—Three distinct forms of lung-syphilis are recognized:

1. *White Hepatization, or White Pneumonia.*—This form is seen in stillborn children with other evidences of hereditary syphilis. The lesion consists in the infiltration and thickening of the walls of the alveoli, the blood-vessels, and the bronchi with small cells. Some of the air-spaces are filled with epithelial cells. These cases are of pathological rather than of clinical interest.

2. *Gummata* may occur in the lung as a late tertiary

manifestation. The gummata are distributed through the lungs and are especially numerous at the bases; they vary from a microscopical size to that of a lemon. Each gumma is usually surrounded by a zone of consolidation. There are usually a general bronchitis, thickened pleura, and some interstitial pneumonia as complicating lesions.

Destructive syphilitic processes in the lung have not as yet been proved. Cases of gummatous pneumonia resemble, clinically, tubercular broncho-pneumonia or new growths of the lung.

3. *Syphilitic Fibroid Pneumonia.*—In this form the pleura is thickened, and bands of connective tissue extend from it into the lung. The bronchi are surrounded by a growth of connective tissue, which may either so compress them as to cause urgent dyspnœa or may weaken them and allow of their dilatation. Patches of indurated connective tissue may replace the lung-parenchyma, rendering certain portions of the lung completely solid. All these lesions are found especially well marked at the base of the lung. The physical signs are those of bronchitis, bronchiectatic cavities, thickened pleura, and areas of consolidation.

The **treatment** is that of tertiary syphilis, but it is usually ineffective. The course of the disease is slow, and the termination is usually fatal.

NEW GROWTHS OF THE LUNG.

The most common forms of neoplasms of the lung are carcinoma, especially of the epithelial variety, and sarcoma; more rarely are found enchrondroma and osteoma. Primary growths are exceedingly rare.

Primary carcinoma is usually found as a single growth at one apex; it may involve other organs by metastasis through the medium of the bronchial glands, or it may involve the pleura and the chest-wall by direct extension.

Secondary carcinoma occurs as scattered nodules through out both lungs, each nodule being surrounded by a zone of congested and consolidated lung-tissue. Either a simple or a malignant pleurisy usually complicates the lesion.

Sarcoma is usually secondary, and both lungs are involved by scattered nodules. The sarcoma is usually of the small-celled variety.

Symptoms.—1. *Symptoms due to involvement of the lungs* depend on the size, number, and position of the malignant growths. If the nodules be small and scattered, the patient will complain of dyspnœa and of pain in the chest from the complicating pleurisy, and will develop a cough. The expectoration is either muco-purulent or bloody, or perhaps dark and mucoid, the so-called "prune-juice" expectoration, which is highly suggestive. In other cases the expectoration resembles currant-jelly, and this appearance is almost pathognomonic. In the expectoration cancer-cells may be found.

2. Large single growths may cause *pressure-symptoms.* Pressure on the bronchi will cause cough, expectoration, hæmoptysis, and dyspnœa. Pressure on the vena cava will cause congestion and œdema of the arm, the neck, and the upper portion of the thorax. Pressure on the œsophagus will cause dysphagia. Pressure on nerves will cause intercostal neuralgia. If the recurrent laryngeal nerve be involved, the patient will develop a brassy cough, laryngeal voice, and steady or paroxysmal dyspnœa.

3. *Cancerous cachexia* will be shown by a waxy pallor, loss of strength, slight afternoon fever, and œdema of the ankles.

4. There may be added the symptoms of primary or secondary growths.

Physical Signs.—Inspection may reveal distended veins of the upper thorax and the neck or bulging of the chest-wall, which may be mistaken for aneurysm, especially as the bulging part may yield a slight expansile pulsation or an appreciable transmitted expansion. There may be infiltrated cervical or axillary glands, and the seat of the primary growth may be detected.

Physical signs depend upon the size and position of the tumors.

1. There may be only the physical signs of a bronchitis with those of a dry pleurisy or of an effusion which in some cases is hemorrhagic.

2. Larger nodules give rise to the signs of scattered areas of consolidation.

3. A single large growth will give rise to an area of flatness, with absence of voice and breathing, surrounded by an area of dulness, with bronchial voice and breathing. The central flat area is usually exquisitely tender on percussion.

The **prognosis** is fatal in from six months to two years.

Treatment is merely palliative, to relieve suffering. Injections of the toxic products of the erysipelas coccus may be made in sarcomatous cases.

ECHINOCOCCUS CYSTS OF THE LUNG.

Small cysts may exist for a considerable time without causing symptoms. Large cysts tend to compress the lung and lead to secondary inflammatory changes.

Rupture into a bronchus will allow of the expectoration of fragments of the cyst-wall and of the hooklets, establishing the diagnosis without doubt.

Rupture into the pleura is not uncommon. Suppuration of the cyst may occur, and gangrene of the lung is not uncommon.

(*b*) Tubercular Diseases of the Lung.

TUBERCULAR INFLAMMATIONS IN GENERAL.

Definition.—Tubercular inflammation, caused by infection by the tubercle bacillus, is characterized by the production of new tissue of low vitality.

Etiology.—The bacillus tuberculosis, first described in 1881 by Koch, is now definitely proved to be the actual cause of tubercular disease. The bacillus is a short fine rod having a length equal to one-half the diameter of a red blood-cell. When stained it presents a beady appearance, probably due to spore-growth within it. For the methods of staining the germ the reader is referred to works on bacteriology. The bacilli are found in all tubercular lesions, but they are more numerous in the acute forms of disease. They may gain access to the blood-vessels or the lymph-vessels, and become generally distributed throughout the body. They are thrown off in the expectoration of patients suffering from pulmonary tuberculosis in enormous numbers, and this infected sputum, when allowed to dry, enters the air as a fine dust, which spreads the disease in every direction and infects rooms, carpets, and clothing.

Modes of Infection.—1. *Hereditary or congenital tuberculosis* is very rare, although undoubted cases have occurred.

2. *Acquired Tuberculosis.*—(*a*) *By Inhalation.*—Inhalation is the most common method of infection, as is proved by the fact that 50 per cent. of all autopsies show some degree of tubercular disease of the lungs. Cloisters, prisons, asylums, and infected houses show a marked increase of tubercular inhabitants. The expired air, however, of tubercular patients is not infective.

(*b*) *By Inoculation.*—Local tubercular lesions may result from inoculation incurred by handling tuberculous pathological specimens, infected meat and skins, and by wounds being inoculated by impure instruments or by tubercular dust. Many cases of tuberculosis in children have followed the rite of circumcision, during which the wound has been sucked by a tuberculous operator. There is no evidence that tuberculosis can be conveyed by vaccination with humanized virus.

(*c*) *By the Ingestion of Tuberculous Meat or Milk.*—Striking examples of the infectious properties of the milk of tuberculous cows are, unfortunately, only too common, many cases of intestinal and mesenteric tuberculosis being directly traceable to this cause. Meat of tuberculous animals is not always infective, and the process of cooking in all probability affords an efficient safeguard against this method of transmission.

Conditions Favorable to Infection.—1. *Constitutional Conditions.*—(*a*) A family history of tuberculosis is present in from 10 to 50 per cent. of cases, according to various authors. It is hard to say, however, in any given case, whether the child was born with some inherent tissue-weakness predisposing toward tubercular infection—a naturally good soil for the growth and development of the bacilli—or whether greater risk for accidental infection was run from the child living with, kissing, and sleeping with tuberculous parents. In either case, however, transmission of the disease is more common when the mother is tuberculous.

(*b*) Tubercular infection is more common among weakly, sickly subjects with deficient chest-expansion. Any depreciation of the general health diminishes the resistance of the tissues and favors infection. It is found that rabbits, by being allowed to run freely in the woods, may be kept in such perfect condition that they cease to be good subjects for the experimental inoculation of the bacilli.

(*c*) No age is exempt, but in children tuberculosis of the bones, the lymphatics, the meninges, and the intestinal tract is much more frequent than in adults.

(*d*) The negro affords a fertile soil for the growth of the bacilli, and tuberculosis among the American Indians is especially frequent.

2. *Local Conditions.*—Any local weakness or inflammation renders the part of the body affected more susceptible to tubercular infection. Bronchitis, enlarged bronchial glands, and interstitial pneumonia are frequently found as predisposing causes of pulmonary tuberculosis, while intestinal catarrh produces conditions favorable for the growth of the bacilli in the alimentary tract. Local injuries or operations may so weaken the tissues as to allow of infection. Thus a simple synovitis from injury may become tubercular, or pulmonary tuberculosis may follow severe contusions of the chest.

Structure of Tubercle.—The local action of the tubercle bacillus upon the tissues results in the proliferation of preexisting cells and in the aggregation of leucocytes. To this collection of cells the name of "tubercle" is given.

The first step in the formation of tubercle consists in the increase in number of the fixed normal cells, especially those of the connective tissue and the endothelium of the capillaries. These proliferated cells are known as "epithelioid cells." Giant-cells are formed by the increase in protoplasm and in the nuclei of a single cell or by the fusion of several cells. Giant-cells are found, however, only in cases in which the bacilli have a low degree of vitality.

The next step consists in the emigration from the neighboring blood-vessels of leucocytes which mix with or surround the above-mentioned epithelioid cells. A network or reticulum of fibres is found between the cells, probably representing the original interstitial tissue stretched apart by the increased cellular elements. The reticulum is most marked at the margin of the tubercle. No new blood-vessels are formed in the tubercular growth, and pre-existing blood-vessels are apt to show lesions of an obliterating endarteritis, so that the newly-formed tissue has but feeble vitality.

When these changes have become sufficiently extensive the tubercle appears as a grayish point or nodule, to which the name "miliary tubercle" is given. By the growth and approximation of these miliary tubercles large areas may be involved, forming the so-called "diffuse tubercle."

Histologically, tubercle cannot always be differentiated from other infectious tumors, such as those of syphilis or leprosy, or from ordinary granulation-tissue. Tubercle-tissue, however, is characterized by its natural tendency toward cheesy degeneration.

Cheesy degeneration, or "coagulation-necrosis," is due partly to the local action of the bacilli upon the newly-formed cells, and partly to the scanty blood-supply of the tubercular nodule. At the centre of the nodule the cells die, lose their nuclei and their staining properties, and become translucent and structureless. This area of degeneration presents an opaque, yellowish-white appearance, and by the extension and coalescence of these areas the degeneration may become exceedingly extensive. Subsequently the degenerated area may undergo (1) *softening*, so as to form a cavity or an ulcer, (2) *encapsulation*, or (3) in it may be deposited the *salts of lime*.

Spontaneous healing of tubercular nodules may occur in two ways:

1. The nodule may be surrounded by a fibrous capsule; its cheesy centre may be converted to a putty-like mass which may be infiltrated with the salts of lime; or it may liquefy and be absorbed, leaving only a puckered cicatrix.

2. There may be a great increase in the fibroid elements of the nodules, so that the tubercle becomes firm, hard, and

does not increase in size. This fibroid or sclerotic change is more frequently seen in tubercles of the peritoneum.

Secondary Inflammatory Processes.—Tubercle seldom occurs alone in the tissues, but the irritation caused by its growth produces secondary inflammatory changes, so that the tubercle may become a very composite structure.

Symptoms of Tubercular Disease.—The action of tubercle bacilli is at first entirely local. The affected area may be small, and the resulting symptoms consequently slight and localized. In other cases the local lesion may spread by continuity, so that large portions of important organs may be rendered unfit to fulfil their functions, with resulting symptoms that are not only local but general. In still other cases the lesion is at first localized, but from softening and breaking down of a tubercular deposit the bacilli may enter a blood-vessel or a lymphatic, enter the general circulation, and set up miliary tubercles wherever they happen to lodge. The symptoms resulting from such a scattering of the lesion are those of an acute infectious disease combined with the local symptoms of tuberculosis in different parts of the body. This form is spoken of as "acute miliary tuberculosis;" it is considered under the heading Infectious Diseases. The danger of this general infection must always be borne in mind in every tubercular inflammation, no matter how localized it may be at the onset.

TUBERCULAR DISEASES OF THE LUNG.

Under this heading may be included—1. Acute pulmonary tuberculosis; 2. Chronic pulmonary tuberculosis; 3. Acute pulmonary phthisis; 4. Chronic pulmonary phthisis.

Acute Pulmonary Tuberculosis.

Etiology.—In this form of disease the bacilli reach the lung either through the inspired air or by being transported by the blood. In the latter case tubercular involvement of the bronchial glands may precede the pulmonary tuberculosis. In other cases some old tubercular process is found to be the source of infection.

Pathology.—Disseminated tubercles are found in part of a lung or scattered throughout both lungs. Each miliary tubercle is surrounded by a slight zone of congested or consolidated air-vesicles, but the greater part of the intervening lung-tissue is comparatively normal, and this condition sharply defines the disease from pulmonary phthisis, in which consolidation of the intervening lung-tissue is an early lesion. The tubercles usually are first found at the apex of one lung, from which point they may spread; or tubercles may be formed at once throughout both lungs in great numbers. There is always a catarrhal inflammation of the finer bronchi of that portion of the lung occupied by the tubercles; in rarer cases the bronchitis is general. Tubercles in or near the pulmonary pleura result in pleurisy either with fibrin or with serous effusion. As the tubercles grow and coalesce, portions of lung may be rendered solid, but this consolidation is always a late manifestation of the disease. From areas of softening breaking into one another small cavities may be formed, and these cavities may be still further increased in size by tubercular ulceration of the bronchi leading into them.

Symptoms.—1. Cases in which tubercles are rapidly formed throughout both lungs and in other parts of the body are really examples of acute miliary tuberculosis; they are considered in the discussion of that disease.

2. The term "pulmonary tuberculosis" is applied more properly to those cases in which tubercles spread from the apex of one lung and are not found in other parts of the body—a purely localized process.

In some cases the disease may develop as a primary infection, while in other cases it complicates some pre-existing tubercular lesion. When the disease is established the symptoms depend upon the extent of lung involved, the bronchitis, and the pleurisy.

The patient has a fever, higher in the afternoon, followed by sweating at night. The heart's action is rapid. Breathing is rapid and often insufficient, and is made more rapid by extension of the tuberculosis or by increase of the bronchitis or of the pleurisy. However rapid the breathing, there is not apt to be much subjective dyspnœa, even though there be marked cyanosis. Cough is a fairly constant symptom, and may be most distressing. The expectoration is muco-purulent and may contain blood. In the sputa the bacilli are usually, but not invariably, present. Loss of flesh and of strength is noted from the first, but it seems to bear no direct relation to the extent of lung involved. There is often developed a peculiar pallor of the skin, resembling that of pernicious anæmia. In other cases the skin is cyanotic.

In some patients the symptoms progress steadily, emaciation becomes extreme, and death results from exhaustion or from secondary involvement of other organs, or the patient may pass into a typhoid state. In other cases the process will apparently stop for a time, with a general improvement of all the symptoms. Once quiescent, the tuberculosis may never again extend, and a permanent cure may thus be affected. In other cases, after an interval the process will again start up and involve fresh areas of the lung, with a return of the old symptoms. In still other cases intercurrent attacks of bronchitis, pleurisy, or pneumonia occur to modify the course of the disease.

The **physical signs** may be described as appearing in three stages :

1. The tubercles are scattered at one apex; there is a localized terminal bronchitis and a dry pleurisy. There is diminished expansion at the apex. The percussion-note may be normal, tympanitic, or slightly dull. Vocal fremitus is usually unchanged. Breathing sounds may be feeble or harsh and puerile, but at this stage no tendency toward bronchial breathing is observed. On auscultation crepitant and subcrepitant pleuritic râles and fine bronchial râles are heard, affording strong presumptive proof of pulmonary tuberculosis when localized at one apex (Fig. 38).

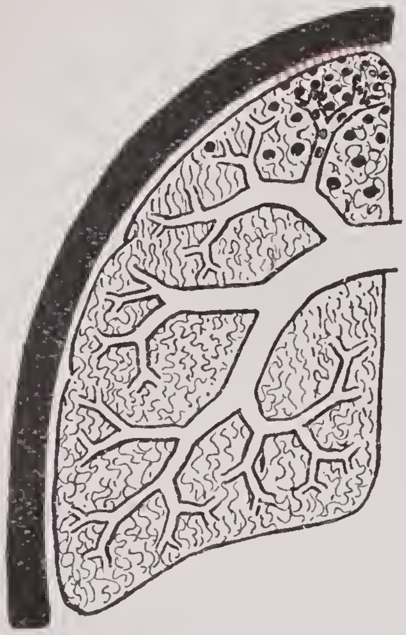


FIG. 38.—Physical signs in the earliest stage of pulmonary tuberculosis: diminished expansion; percussion normal or tympanitic; breathing weak or harsh; pleuritic or bronchial râles.

2. As the tubercles grow and coalesce, portions of lung—usually one or both apices—are rendered solid. Over these portions the note becomes dull, the expiratory sound becomes prolonged and high-pitched, and ultimately bronchial breathing and bronchophony are developed. The physical signs noted in the preceding paragraph spread at the periphery of the lesion (Fig. 39).

3. When small cavities begin to be formed, gurgles make their appearance. The cavities are rarely large enough to yield other and more distinctive signs, although a number of small cavities may merge into one large antrum over which can be obtained a tympanitic or a "cracked-pot" note with broncho-cavernous or cavernous breathing (Fig. 40).

The **diagnosis** of pulmonary tuberculosis should be suspected in every case of dry pleurisy at the apex or of bronchitis at the apex. The disease should be suspected in every case of severe recurrent bronchitis or dry pleurisy, if the constitutional symptoms be out of proportion to the apparent lesion. It should be suspected in the case of any patient with fever and rapid pulse without apparent cause, even in the absence of definite pulmonary symptoms. The positive diagnosis, however, can only be made by the finding of tubercle bacilli in the sputa.

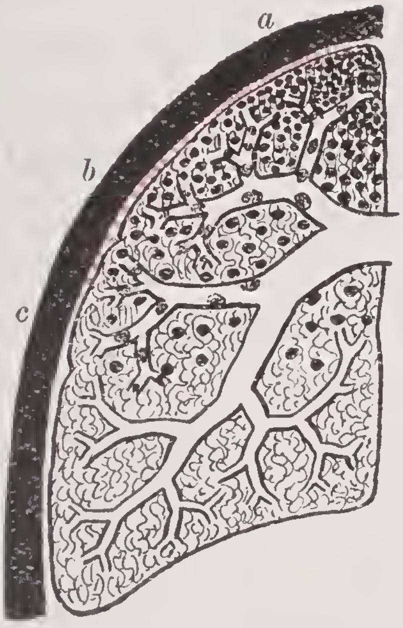


Fig. 39.—Physical signs of pulmonary tuberculosis in the advanced stage : *a*, diminished expansion, dulness, bronchial voice and breathing, increased vocal fremitus; pleuritic and bronchial râles ; *b*, dull tympanitic note, prolonged and high-pitched respiration, bronchial and pleuritic râles ; *c*, tympanitic or normal note, feeble or harsh breathing, pleuritic and bronchial râles.

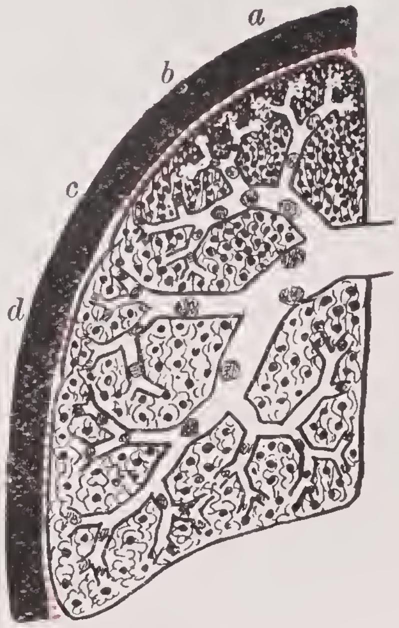


Fig. 40.—Physical signs of pulmonary tuberculosis in the final stages : *a*, signs of consolidation, gurgles, and pleuritic râles ; *b*, signs of consolidation and râles; *c*, dull tympany, prolonged, high-pitched expirations, râles ; *d*, tympany, feeble breathing, râles.

The **prognosis** of rapidly-spreading tuberculosis is bad. Under proper environment the disease may become chronic or may even become quiescent.

Treatment of all tubercular diseases of the lung will be considered later (see p. 395).

Chronic Pulmonary Tuberculosis.

Chronic pulmonary tuberculosis differs from the acute form in the chronicity of its course and the slow reactive nature of the secondary inflammatory processes. Three distinct clinical groups may be described:

I. Miliary tubercles are formed at one apex and slowly spread downward. The opposite apex is next involved in like manner. When the disease is fully developed the tubercles are found scattered throughout both lungs. The miliary tubercles run a very inactive course, some becoming cheesy, while others undergo fibroid change or are encapsulated. There is a chronic bronchitis, at first limited to the apex, later becoming general. Bronchiectatic cavities may form, but they are of rare occurrence. Dry pleurisy, resulting in pleural adhesions and thickening, is due to the irritation of tubercles near or in the pulmonary pleura, and it is usually best marked at the apex. In the later stages of the disease the tubercles may become so closely aggregated as to cause partial or complete consolidation of portions of the lung, especially at the apices; while the formation of small cavities occurs in the most advanced cases.

Symptoms begin insidiously. The first complaint may be of a hacking cough with scanty expectoration, due, as proved by physical examination, to a localized bronchitis at one apex. In other cases a dry pleurisy at one apex will cause pain to be the initial symptom. Other patients suffer from hæmoptysis while in apparently robust health. In other patients the lesions give no subjective symptoms, and the presence of the disease is only discovered by a routine examination of the chest. When the disease has once developed there is a cough, usually troublesome and persistent. It may be dry and hacking or loose in character. Expectoration is scanty at first, but later becomes profuse, muco-purulent, contains bacilli, and frequently is tinged with blood. Hæmoptyses may occur from time to time, but they are rarely profuse during the early stages of the disease.

Dyspnœa is present according to the extent of lung involved, the bronchitis, and the pleurisy. At first noticed only on exertion, it may subsequently become steady and distressing. Extension of the pleurisy is marked by fever and pain. Rapid pulse is almost constant throughout the disease, so that pulmonary tuberculosis should be suspected in all patients whose pulse is continually rapid without apparent cause. Increased rapidity of the pulse and some afternoon rise in fever are caused by fresh accessions of tubercles or by exacerbations of the pleurisy or the bronchitis.

The patient loses flesh and strength, becomes anæmic, and is converted to a semi-invalid. Later in the disease, when small cavities form, "hectic" develops—the afternoon fever, bright eyes, flushed cheeks, night-sweats, and more rapid emaciation.

The **physical signs** of chronic pulmonary tuberculosis resemble those of the acute form. There may at first be no physical signs, or there may be diminished expansion, tympany or tympanitic dulness, with feeble or harsh breathing at an apex. Auscultation reveals pleuritic and bronchial moist râles. In other cases thickened pleura at an apex yields dulness and diminished voice and breathing. Later appear the signs of partial or complete consolidation—dulness, bronchial voice and breathing, and increased vocal fremitus with a persistence of the bronchial and pleuritic râles. The final stage of small cavities is accompanied by gurgles, more rarely by tympanitic or "cracked-pot" percussion-note and by broncho-cavernous or cavernous voice and breathing.

Course of the Disease.—(*a*) In some patients the lesion begins at one apex, but after a certain time does not progress, and the patient lives for years without further trouble. The tubercles still remain in the apex of the lung, but they become encapsulated, fibroid, or calcareous. Many of these patients suffer so little from their disease that no suspicion of tuberculosis is entertained. There is always danger that at any time the process will start anew or be the cause of an acute general miliary tuberculosis.

(*b*) In other patients the lesion is progressive only at long intervals. Exacerbations of the lesion are most apt to occur during the winter months, during any intercurrent pulmonary disease, or whenever the general health is allowed to deteriorate. Many old cases of tuberculosis are thus stirred into renewed activity by epidemics of the "grippe." So a great many patients go on for years, each succeeding exacerbation becoming more and more severe, until finally the extension-process becomes continuous. Under proper environment, however, the course even of these patients may be prolonged and the progress of the disease may ultimately be checked.

(*c*) In other patients the course of the disease is continuous. Pulmonary and hectic symptoms become increasingly pronounced, and death finally results from emaciation, from acute general miliary tuberculosis, from hemorrhage, or from secondary tubercular inflammations of the larynx, the intestines, or the peritoneum.

II. Before the time of the tubercular infection the patient has suffered from emphysema and chronic bronchitis, possibly with the addition of thickened and adherent pleuræ. Such a complex pulmonary condition seems to afford a fertile soil for the development of the tubercle bacilli, and the lesions of pulmonary tuberculosis become combined with the pre-existing morbid changes. The patient at first complains only of the symptoms of the original disorders, but is evidently more seriously sick than he should be with emphysema and bronchitis alone. Gradually the more characteristic symptoms of tuberculosis appear, and the regular physical signs are obtained. The course of this set of clinical cases resembles that of the first variety, but is much more serious, and recovery can hardly be expected.

III. The third set of cases resembles either of the preceding forms except that there is added an interstitial pneumonia. The connective tissue may be arranged around the tubercles, along the bronchi, may extend inward from the pleura in broad bands, or may occur diffusely, rendering parts of the lung completely solid and fibrous. As the result of the interstitial pneumonia the walls of the bronchi become weakened and bronchiectatic cavities are permitted. By suppuration or tubercular ulceration of the walls of the dilated bronchi the bronchiectatic cavities are still further increased in size. The interstitial pneumonia may precede or follow the deposit of tubercles in the lung.

The **symptoms** are more severe than those of the two previous forms, resembling those of chronic pulmonary phthisis. The bronchitis, however, is more extensive; the expectoration is more abundant and may be significant of bronchial dilatation; the dyspnœa is more distressing. Hectic is present in the majority of cases, and repeated small hæmoptyses may occur from the suppurating or ulcerating mucous membrane lining the bronchiectatic cavities. If the interstitial tissue be abundant there will be offered resistance to the flow of blood through the lungs, with hypertrophy of the right ventricle and the symptoms of its dilatation in the latter stages of this disease. To this form of "pulmonary tuberculosis" the term "fibroid phthisis" is often applied.

The **prognosis** of pulmonary tuberculosis is generally better than that of phthisis. The association of emphysema or of interstitial pneumonia with the deposition of tubercles renders the prognosis more serious than that of uncomplicated tuberculosis, not only because the lung is further disabled, but because of the additional danger of failure and dilatation of the right ventricle.

The **treatment** of all tubercular inflammations of the lung will be considered later (see p. 395).

ACUTE PULMONARY PHTHISIS.

Synonyms.—Acute consumption; Galloping consumption; Phthisis florida.

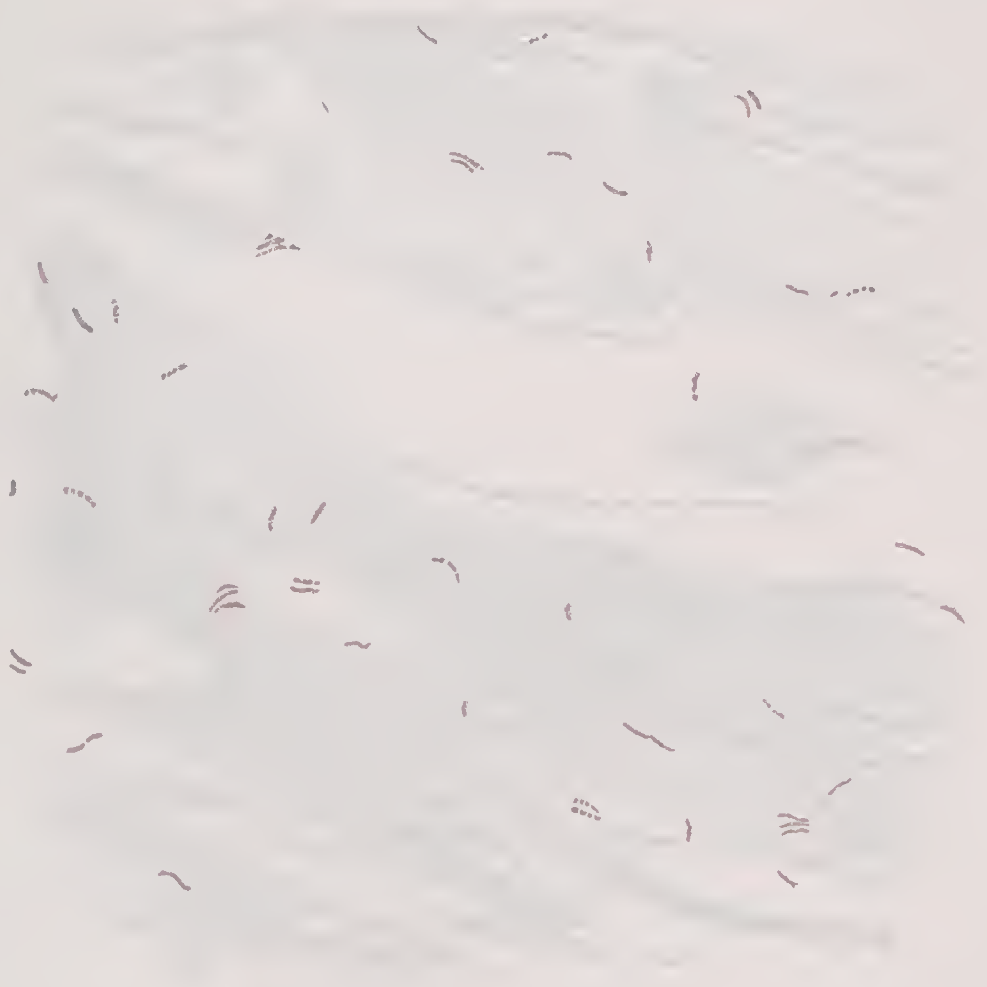
Acute pulmonary phthisis is a much more complex disease than tuberculosis, because other inflammatory changes are mixed with the tubercular lesions in the lungs. Tuberculosis means tubercles alone; phthisis means tuberculosis plus lobar pneumonia or broncho-pneumonia.

Etiology.—Phthisis may be a primary disease or may follow some previous tubercular inflammation of the lung. A person predisposed to tuberculosis will develop phthisis when exposed to the cause of inflammation of the lung with infection at the same time by the tubercle bacilli.

Pathology.—The process usually starts at the apex of one lung and involves the lobe or even the entire lung. In other cases the process may be more evenly distributed throughout both lungs. The affected portions of the lung are *consolidated* from gray or red hepatization or from *yellowish nodules.* There are frequently cavities. The bronchi are inflamed and bronchiectasis may occur. There is fibrin in the pulmonary pleura.

It seems better to describe separately (1) the yellowish nodules, (2) the pneumonia, (3) the lesions in the bronchi, and (4) the cavities.

1. The *yellowish nodules* vary in size from that of the head of a pin to that of a goose-egg. By the coalescence of these nodules still larger nodules may be formed. These nodules consist of tubercle-tissue in the condition of coagulation-necrosis. The tubercle-tissue may fill the air-spaces



Tubercle bacilli in the sputum, first colored with anilin-fuchsin and then with methylene-blue; x about 1000 (Vierordt).

and the small bronchi, being grouped like the inflammatory products of lobar pneumonia; or it may be grouped in the air-vesicles around a bronchus whose wall is infiltrated with tubercle-tissue, in this case resembling the grouping of the inflammatory products of broncho-pneumonia. These nodules are practically composed of dead tissue, and can never be recovered from. They may undergo softening and form large cavities.

2. The *pneumonia* surrounds the nodules and involves the lung-tissue between them. The hepatization may be red or gray in color. In some cases the pneumonia products fill the air-vesicles of a lobe or of the greater part of a lobe, resembling lobar pneumonia. In other cases the grouping is that of a broncho-pneumonia: the pneumonic products occur in patches, each one of which consists of a bronchus with infiltrated walls surrounded by a zone of consolidated air-cells. By the possible coalescence of these peribronchitic patches large areas of lung are rendered solid. The products of either form of pneumonia are perfectly capable of resolution, so that the lung can again return to a healthy condition.

3. The *bronchi* may be the seat of a catarrhal inflammation, or their walls may be infiltrated by the products of tubercular or non-tubercular inflammation. Infiltration of the bronchial wall weakens it, allowing of cylindrical or sacculated dilatation. If the infiltration be tubercular, ulceration of the bronchial wall may occur, still further increasing the size of the bronchiectatic cavity.

4. *Cavities* may be formed in several ways: (*a*) By the breaking down of tubercular nodules; (*b*) by bronchial dilatation; (*c*) by ulceration of the walls of the bronchi.

Grouping of the Lesions.—Delafield describes three distinct types of phthisis, having the same clinical history, but differing in appearance and in the physical signs:

1. One or more lobes are completely consolidated by the filling of the air-spaces and the small bronchi with epithelium, fibrin, and pus. Scattered throughout the consolidation are tubercular nodules. The pleura is coated with fibrin. This form of phthisis is described as "acute pneumonic phthisis" or "acute tubercular pneumonia."

2. There is a general catarrhal bronchitis, and a tubercular inflammation of the walls of some of the bronchi and of small zones of air-spaces surrounding them. This form of phthisis is spoken of as "tubercular broncho-pneumonia."

3. The third type resembles the preceding form except that in addition to the tubercular broncho-pneumonia there are large or small areas of diffused consolidation due to the filling of the air-spaces with fibrin, pus, and epithelium.

Cavities and bronchiectasis may occur in any one of the preceding types.

The **symptoms** of phthisis begin acutely or gradually. If the onset be *sudden*, the symptoms resemble those of pneumonia. There is a chill followed by fever, pain in the side, a cough with mucous or muco-purulent expectoration, and much prostration.

The differential diagnosis of these cases from pneumonia is always difficult, and during the first days of the disease it may be absolutely impossible. Pneumonia, however, defervesces between the sixth and the twelfth day, with improvement of all symptoms, whereas in phthisis the patient's condition becomes aggravated, night-sweats appear, and the expectoration is more purulent and profuse. Even then the case may resemble one of tardy resolution. In pneumonia hæmoptysis does not occur; in phthisis it may be an early symptom. The fever in phthisis is more remittent than in pneumonia. Should cavities form or bacilli be found in the sputum, the differential diagnosis would present no difficulties.

If the onset be *gradual*, there will be a cough, dry at first, later becoming moist and accompanied by a muco-purulent expectoration which may contain blood. Hæmoptysis in considerable amounts may occur. There is a fever of from 100° to 102° F. with afternoon exacerbations. The breathing is rapid, but subjective dyspnœa is seldom distressing. When the disease is developed the cough becomes looser; the expectoration becomes more abundant and more purulent and contains the bacilli. Large hæmoptyses arise from ulcerated arterial branches in the walls of cavities; repeated small hemorrhages arise from the ulcerating walls of bronchiectatic cavities.

The fever is usually remittent—about 100° F. in the morning, with an afternoon exacerbation to 103° or 104° F. The remissions occur regularly in the early morning hours; they are accompanied by profuse cold sweats, especially about the head and the neck. During the exacerbation of the fever the cheeks are flushed, the eyes are bright, and the whole demeanor of the patient is cheerful throughout. The pulse becomes increasingly rapid and feeble; the breathing becomes more and more rapid, and it may be so inefficient that cyanosis becomes marked, but there is rarely any complaint of dyspnœa. The patient rapidly loses flesh and strength and becomes anæmic.

The **physical signs** at first are those of broncho-pneumonia or of lobar pneumonia. Over the consolidated portions of the lung there are diminished expansion, dulness on percussion, bronchial breathing with bronchophony, and subcrepitant and crepitant râles. If there be a tubercular broncho-pneumonia without much consolidation, there may be only sibilant and sonorous breathing with bronchial râles, or small areas over which there are dulness on percussion, intensified voice, and crepitant and subcrepitant râles. When cavities form there is obtained tympanitic dulness or a "cracked-pot" or even an amphoric note; the breathing and the voice become cavernous, and gurgles and churning râles make their appearance. In cases in which the cavities are full of secretion there may be flatness and absence of voice and breathing, but moist râles of all kinds are heard with both inspiration and expiration.

Course of the Disease.—(1) Some patients die in two or three weeks in a typhoid condition or from exhaustion or hemorrhage. The diagnosis of lobar pneumonia is often made in these cases. (2) In other patients the disease is prolonged for several months. (3) Some patients pass into the condition of chronic phthisis. (4) In a small minority recovery may occur; the tubercular products are encapsulated or undergo fibroid change, while the non-tubercular products undergo resolution.

The **prognosis** of acute phthisis is unfavorable. Recovery is rare, but in a large number of patients the lesions become chronic, so that life may be considerably prolonged.

The **treatment** of acute cases resembles that of pneumonia; in subacute cases the treatment is that of chronic pulmonary phthisis.

CHRONIC PULMONARY PHTHISIS.

Etiology and Synonyms.—Chronic phthisis may appear as a primary form of disease or may be secondary to pulmonary tuberculosis or to any pre-existing tubercular disease of the lung. The chronic form may follow acute phthisis or may be chronic from the start. *Synonyms:* Chronic consumption; Chronic ulcerative phthisis.

Pathology.—The lesions of chronic phthisis resemble those of the acute form, except that they are modified by their long duration and that there is added an interstitial pneumonia. Separate descriptions may be given of (1) the tubercular nodules, (2) the pneumonia, (3) the interstitial pneumonia, (4) the changes in the bronchi, (5) the cavities, and (6) the lesions in the pleura.

1. The *tubercular nodules* are arranged as are those of acute phthisis. In some cases the air-spaces are filled with tubercle-tissue; in other cases peribronchitic areas of tubercular consolidation occur, the wall of the central bronchus being infiltrated with tubercle-tissue. The tubercular nodules may undergo coagulation-necrosis or may break down, forming cavities. In favorable cases the nodules undergo fibroid change or become encapsulated with cheesy or calcareous centres.

2. The products of *pneumonia* are grouped like those of the lobar form, the air-spaces being filled with fibrin, pus, and epithelium, or there may be the peribronchial nodules of a broncho-pneumonia.

3. The *interstitial pneumonia* develops in several ways: (*a*) There may be areas of lung-tissue converted more or less completely to fibrous masses. The walls of the air-spaces are thickened; their cavities are encroached upon by polypoid outgrowths of connective tissue, and they may be filled with epithelial cells. (*b*) Bands of connective tissue extend along the bronchi, the blood-vessels, and the septa between the lobules. (*c*) Bands of connective tissue penetrate the lung, extending inward from a thickened and adherent pleura.

4. The *bronchi* undergo the same inflammatory changes as in acute phthisis: (*a*) They are the seat of a chronic catarrhal inflammation. (*b*) Their walls may be infiltrated by the products of a tubercular or a non-tubercular inflammation, and, being thus weakened, sacculated bronchial dilatation will result. (*c*) If the infiltration be tubercular, ulceration of the bronchial wall will further increase the size of the bronchiectatic cavity.

5. *Cavities* are formed—(*a*) By the softening and break-

ing down of areas of coagulation-necrosis; (*b*) by bronchiectasis; and (*c*) by tubercular ulceration of the walls of the bronchi. Cavities, when once formed, tend to enlarge, and as they increase in size they touch and open into one another. In this way the greater part of a lobe may be converted to a single large cavity. A blood-vessel is the last structure to be ulcerated in the formation of a cavity. An obliterating endarteritis usually occurs, converting the vessel to a fibrous cord, thus preventing hemorrhage at the time of its erosion. Should this conservative process not be completed, partial erosion of the arterial wall will allow of the formation of a little aneurysm, which may finally rupture, causing profuse hemorrhage. Conservative and healing processes may occur even after a cavity has once formed. The wall of the cavity becomes thick and fibrous, and the lining may be smooth, resembling mucous membrane. Healing processes, however, do not occur in cavities of any size.

6. The *pleura* over the involved area of lung is regularly the seat of a chronic inflammation, resulting in thickening and adhesions. This form of chronic inflammation is really conservative, as, by the strengthening of the pleura by connective tissue the risk of perforation of cavities or of bacterial infection of the pleura is minimized.

The lesions of chronic phthisis usually begin at the apex of one lung and extend downward to involve the upper lobe and the apex of the lower lobe. The opposite apex is then regularly affected.

Complicating and secondary lesions will be considered under the heading "Complications."

Symptoms.—The mode of onset is varied and insidious.

1. The disease may begin with dyspeptic and anæmic symptoms not readily alleviated by treatment. Amenorrhœa is an early symptom of these anæmic patients.

2. There may be a gradual loss of flesh and of strength, with a slight afternoon rise in temperature.

3. The symptoms of a "neglected cold" may precede other symptoms. Cases of recurring or of persistent bronchitis, especially in a young person, should always be regarded with suspicion.

4. Chills and fever due to tubercular infection may be mistaken for those of malarial origin.

5. The disease may begin with a laryngeal cough and huskiness of the voice, and on examination either a catarrhal or a tubercular laryngitis may be found, or the larynx may appear simply anæmic.

6. Hæmoptysis may be the initial symptom, preceding other manifestations of the disease by months or even by years.

7. There may be dry pleurisy, especially at an apex or in the scapular region, or a pleurisy with effusion running an acute or an insidious course. According to Bowditch, phthisis ultimately develops in one-third of the cases of pleurisy with effusion. A double pleurisy with effusion is much more suggestive of tubercular origin.

Symptoms of the Developed Disease.—1. *Pulmonary Symptoms.*—(*a*) Cough is an early and almost a constant symptom. Dry and hacking at first, it later becomes looser and more frequent. It may be so distressing as to prevent sleep, and sufficiently severe and paroxysmal to provoke vomiting and thus to interfere with the patient's nutrition. (*b*) The sputum varies in amount and character in the different stages of the disease. At first the expectoration is mucous and of a glairy consistency, presenting nothing suggestive of tubercular trouble. Later in the disease the sputum becomes muco-purulent and contains little grayish or grayish-green lumps. When cavities form the expectoration is more profuse, especially in the morning or after sleep, is more purulent, and finally the sputa assume the nummular form of separate solid purulent masses which sink in water. The expectoration of phthisical patients has usually a heavy sweetish odor, although it may be fetid. In cases of consolidation without much bronchitis the sputum may not be abundant at any time. Generally the quantity of the sputum gives a fair test of the activity of the disease. Examination of the sputum for tubercle bacilli should always be made in doubtful cases. The bacilli are usually present early in the disease; they are abundant in proportion to the intensity of the tubercular process. A diminished number of bacilli affords grounds for a more favorable prognosis. The presence of bacilli in the sputum is an infallible proof of the existence of tuberculosis, but their absence does not necessarily exclude the disease. Tuberculosis can be excluded only after repeated examinations of the sputa show absence of the bacilli.

The demonstration of elastic fibres in the expectoration only proves the existence of some destructive pulmonary lesion, the fibres being found in tuberculosis, gangrene, and abscess of the lung. If the sputum be pressed between two thin cover-glasses and held against a black ground, the elastic fibres can usually be recognized with the naked eye. From the appearance of the elastic fibres it can be told whether they are derived from the bronchi, the alveoli, or the blood-vessels.

Hemorrhage occurs in 60 per cent. of all cases of chronic pulmonary phthisis; it may appear early or late in the disease. Large early hemorrhages never lead to phthisis, as is erroneously supposed, but arise from a small undiscovered lesion. The small early hemorrhages usually arise from the congested or ulcerated walls of the bronchi, and the blood is admixed with sputum. Large late hemorrhages arise from the erosion of an artery or from a ruptured aneurysm of an artery within a cavity; in these cases the blood is profuse and is unmixed with sputum. For the differential diagnosis of hæmoptysis from hæmatemesis see Pulmonary Hemorrhage.

Small hemorrhages from congested bronchi may relieve congestion and may be followed by a feeling of general improvement. Large hemorrhages are often fatal, either from the exhaustion and anæmia induced by them, or by reason of the hemorrhage itself, or because blood is aspirated into the bronchi of the other lung, causing asphyxia or septic pneumonia.

Pain may be a distressing symptom or it may be absent entirely. When present it is due either to the pleurisy, to the muscular strain of coughing, or to intercurrent intercostal neuralgia.

Tenderness is often elicited by percussing over the locality of a dry pleurisy.

Dyspnœa on exertion increases with the extension of the disease and with the exacerbations of the bronchitis. Constant dyspnœa usually indicates excessive involvement of both lungs or points to some pleural complication. Extreme dyspnœa with cyanosis is practically unknown in uncomplicated cases. Pain and sudden urgent dyspnœa suggest pneumothorax.

Constitutional Symptoms.—Fever usually is marked in proportion with the advance of the lesion, and a persistently normal temperature usually means that the disease is not progressing. It is possible, however, for consolidation alone to cause no fever, the febrile condition in general being due to the bronchitis and to the suppuration in the cavities. The fever is usually remittent or even intermittent, the minimum temperature occurring between 2 and 6 o'clock A. M., the maximum being noted between 2 and 6 o'clock P. M. The afternoon rise of temperature is usually accompanied with flushed face, brilliant eyes, and a "hectic flush." The early morning remission is marked by profuse cold night-sweats, especially about the head and the neck. The sweating in advanced cases also recurs during the day, after sleeping. When extensive suppurating cavities exist the morning temperature may be subnormal. A continuous high temperature suggests an intercurrent pneumonia. The temperature is often influenced by rest and by good nursing, and usually declines with hospital care. The temperature becomes also less marked under favorable changes of climate.

The pulse is rapid, full, and compressible, and there may be capillary pulsation visible under the finger-nails.

The patient loses flesh and strength and becomes anæmic. These changes depend upon the fever, the progress of the disease, and the proper feeding and treatment of the patient. The weight, which gives a good index of the progress of the disease, should always be considered in the prognosis. It is possible, however, for the patient to retain flesh, strength, and color even with a well-advanced lesion.

The mental state is peculiarly cheerful, and even moribund patients are firmly confident of a speedy recovery.

Menstruation in women becomes irregular or ceases altogether.

Digestive Symptoms.—*Anorexia* may be a well-marked symptom, so that there is actual loathing for all food. *Nausea and vomiting* may appear in the later stages, being due to paroxysms of coughing or to dilatation or a chronic catarrhal inflammation of the stomach. As a rule, however, phthisical patients digest well, although the stomach lacks its normal peristaltic power and the gastric juice is deficient in HCl.

Diarrhœa often appears in the later stages of the disease; it may be due to waxy or fatty degeneration of the liver, to catarrhal enteritis, to amyloid degeneration, or to tubercular ulcerations of the intestine, especially of the large bowel. Tubercular ulceration of the ileum may cause no diarrhœa, but it induces an emaciation that cannot otherwise be accounted for. In some cases no lesion is found post-mortem to account for the diarrhœa.

Physical Signs.—1. *Signs of Early Cases.*—There is usually appreciated by palpation a diminished respiratory expansion at one apex; this sign, which often precedes all other physical signs, is of great diagnostic importance. The percussion-note over and under the clavicle may be normal or slightly dull. Breathing (1) may be simply feeble, or (2) the inspiration may be inaudible, while the expiration is unduly prolonged, or (3) the respiratory murmur may be harsh and rude and of the peculiar wavy, jerky character spoken of as "cog-wheel" breathing. Fine moist bronchial râles and subcrepitant pleuritic râles are frequently heard even in early cases.

2. *Signs of Evident Consolidation.*—The deficiency of local chest-expansion becomes more marked, and inspection may show some sinking of the infraclavicular spaces. The percussion-note is slightly dull; the breathing and the voice approach the broncho-vesicular (louder and higher piched than normal, with an expiration longer and higher in pitch than inspiration). Vocal fremitus is usually increased unless there be thickened pleura. These physical signs are distinctive when obtained at the left apex, but are nearly the signs normally obtained at the right apex; the presence of bronchial and pleuritic râles, however, not being normal to either apex, may make the diagnosis evident. Later, when consolidation becomes more marked, the dulness becomes more pronounced, the breathing and the voice become bronchial, vocal fremitus is increased, and the bronchial râles become coarser and more numerous.

3. *Signs of Cavities.*—Numerous scattered cavities without much surrounding consolidation and without pleuritic thickening may yield a nearly normal percussion-note. On auscultation, however, bronchial breathing and gurgles are heard. Small cavities filled with secretion may give rise to marked dulness or even flatness. Tympany is excited over cavities of about the size of an English walnut. The tympanitic quality is best marked when the patient's mouth is open, constituting "Wintrich's sign." A "cracked-pot" note is obtained by firm, sharp percussion over superficial cavities having yielding walls, with open communication with a bronchus. This note often comes and goes, is reproduced by coughing, and is best heard when the percussor's ear is placed directly in front of the open mouth of the patient. It must be remembered that a "cracked-pot" note may be normally heard at the right apex in some children. An amphoric note is heard by percussing large cavities with smooth rigid walls.

The breathing over cavities may be bronchial, bronchocavernous, or cavernous. The breathing signs, however, change their character according to whether the cavity is empty or is filled by secretion. A distinctive form of breathing heard over cavities consists of a respiratory murmur, beginning as vesicular and suddenly breaking into bronchial.

Gurgles and churning sounds are heard over most cavities, but there are exceptions in which the cavities are dry.

It should never be forgotten that the signs of a cavity may be simulated exactly by a patch of consolidation over a large bronchus, so that the diagnosis of a cavity should always be made with extreme caution.

Complications.—There may be pleurisy with effusion or empyema. Perforation of the pleura over a softening tubercular nodule results in pneumothorax or of pyo-pneumothorax. There may be tubercular inflammation of other organs, especially meningitis, tubercular laryngitis, and ulcerations of the intestine. The kidneys may be tubercular or may be the seat of amyloid change. Chronic diffuse nephritis may develop. The liver may be waxy, fatty, or the seat of tubercular deposits. Tubercular peritonitis may be present, or septic peritonitis may result from the rupture of tubercular intestinal ulcerations. Phthisis at any time may be complicated by pulmonary tuberculosis or by acute general miliary tuberculosis. Lobar pneumonia is not uncommon as a terminal event. There may be developed in phthisical patients a form of insanity resembling that occurring during the convalescence from acute diseases. Peripheral neuritis is occasionally observed.

The **prognosis** is grave, but not hopeless. Favorable results are common in early cases properly treated by change of climate, while spontaneous cures are not infrequent, even under unfavorable hygienic surroundings. The prognosis is dependent upon the constitutional vigor and the financial condition of the patient, and the rapidity of growth and the extent of the lesions. Repeated hæmoptyses are unfavorable.

Treatment of Tuberculosis in General.

There are three indications for treatment: (1) To prevent the spread of the disease; (2) to arrest the disease; and (3) to relieve symptoms.

1. *Prophylactic Treatment.*—(*a*) *For the General Public.*— The sputa of all tubercular cases should be collected and destroyed. The patient should be warned not to spit about the house or in the street. Portable spit-cups are invented for the collection of the sputa; or handkerchiefs may be used for the purpose if they are thoroughly boiled, after use,

in a receptacle separate from that for the other clothes. A phthisical patient should sleep alone, and separate staterooms on steamships should be provided for tubercular cases. Rooms infected by tubercular sputa should be disinfected thoroughly before they are again occupied. There should be governmental inspection of dairies and slaughter-houses, and tuberculosis in animals should be stamped out by killing the infected animals.

Patients with tuberculosis should not marry. In women with a suspected tubercular tendency the risk of developing the disease is largely increased by childbearing.

(*b*) *For the Individual.*—A child born of tuberculous parents should receive careful prophylactic treatment. The mother of a tuberculous child should not nurse it nor sleep in the same room with it. The child should enjoy the recreations of an outdoor life, and studious habits, especially in crowded schools, should be sacrificed for athletic pursuits. Sedentary occupations are undesirable. Nasal obstruction, enlarged tonsils, and adenoid disease, if present, should receive prompt attention, so as to allow of the fullest extent of breathing. All intercurrent diseases are to receive more than usual attention, and the general health is to be kept at the highest pitch by fresh air, sufficient sleep, proper food, and tonics whenever they may be required.

2. *To arrest the disease* two things are necessary—to keep the general health good, and to prevent complicating inflammations. These conditions are complied with by (*a*) climate, (*b*) hygiene, (*c*) diet, and (*d*) drugs.

(*a*) Suitable change of climate affords the best chance for permanent recovery. Usually, however, the change is insisted on too late, and hopeless, even dying, patients are sent on long journeys away from friends and home comforts. There is no one climate suitable for all tuberculous patients, and in the selection of a climate good judgment and common sense must be employed. Generally speaking, the requirements are a pure atmosphere, an equable temperature, and a maximum of temperature. As to the exact choice, much depends upon the patient. Young and robust patients with early lesions do best usually in a cold, bracing climate, where they can lead an active outdoor life and become strong and muscular. Such cases do well in the Adirondacks or in Colorado. Those who are unable to take physical exercise by reason of age, sex, or advanced pulmonary lesions do best in a warm, dry, equable climate, where they can sit outdoors and keep from catching cold without being obliged to exercise. Such a climate is to be found in Southern California, North and South Carolina, Georgia, Florida, Mexico, Egypt, and Algeria. The more unable such patients are to exercise, the warmer the climate they seem to need. Some patients are rendered worse in the cool climates and are debilitated by warm air. Such patients should travel from place to place until they find a climate in which they improve in one or two weeks. Other patients seem to do best by a variety of climates, and they improve by continually travelling.

(*b*) *Hygiene.*—Tubercular cases require a maximum of fresh air and sunshine. The sleeping-room should be airy and sunny. Exercise in the open air should be graded to the strength of each patient, much harm being done by conscientious exercise past the point of moderate fatigue. The skin should be kept open by skin-frictions and daily baths. The patient should avoid exposure to inclement weather, but over-coddling weakens him and increases his liability to catch cold. The patient should wear flannels throughout the year, but should not be over-clothed, as the danger of catching cold is thereby increased.

(*c*) *The diet* should be simple, wholesome, and abundant. The rule is that tubercular cases should be over-fed. In addition to the ordinary diet, as much milk and cream as possible should be taken, and all dyspeptic symptoms should receive proper attention. If milk and cream are not well borne, cod-liver oil should be given in as large doses as the patient will tolerate. Superalimentation by the stomach-tube is often of great benefit. Alcohol with meals may be allowed if it agrees.

(*d*) *Drugs.*—A large number of specifics are lauded every year, but each one fails in fulfilling expectations. There is no specific treatment. A glycerin extract of the culture of the tubercle bacilli was first used by Koch, and it was found to exert a specific effect on tubercular inflammations. Injections of one milligram were followed by intense constitutional and local reaction, and cures of external tuberculosis, such as lupus, were recorded. In internal tuberculosis, however, old quiescent lesions were stirred into activity, and acute miliary tuberculosis often developed from a local lesion. Various modifications of Koch's lymph have been used, but they should be used with extreme caution. At present the feeling among the profession is strongly against the use of the lymph, but modifications and improvements may in the future place it among the standard list of specific drugs.

Creosote, which has been gaining steadily in favor, is one of the best remedies in use. It may be given in 4-minim doses at first, gradually increased to 10 or 15 minims three times a day. It may be given with compound tincture of gentian or with glycerin and whiskey, and it should be diluted largely with water at the time of its administration, or it may be given in capsules. Only the pure beechwood creosote should be prescribed. The "enteric pill" of Parke, Davis & Co. contains creosote; it is not dissolved until it reaches the small intestine. The patient may wear continuously a perforated zinc inhaler (Robinson's) kept moistened with equal parts of alcohol, chloroform, and creosote. Creosote may also be given by the rectum, from 5 to 20 drops being mixed with the white of one egg and water and given every day. Guaiacol or the carbonate of creosote may be used in substitution. Iron, strychnine, and arsenic are useful tonics in combination, to combat the anæmia and to build up the general strength. The hypophosphites are useful tonics, but they have no specific action.

Injections of antiseptics into the diseased pulmonary tissues have been advocated warmly; they are not commonly employed, however, as bad results have occasionally followed their use.

In some cases the inhalation of compressed air has been of great service.

3. *To Relieve Symptoms.*—Fever, as a rule, is best treated by change of climate and by fresh air. When the temperature is high, however, patients should not attempt much exercise, and frequently they do better when put to bed for a few days. Sponging with cool water relieves the feverishness and makes the patient comfortable, but other more radical measures are to be used with extreme caution.

For the sweating, aromatic sulphuric acid is the best and the simplest remedy. Sponging the body with vinegar and water upon retiring is frequently effective. Zinc oxide, gr. ij, with ext. hyoscyami, gr. iij, in pill is a favorite combination, while atropine in doses of gr. $\frac{1}{120}$ at night is fairly steady in its effects. Picrotoxin (gr. $\frac{1}{60}$) may be used, but with extreme caution. Strychnine is often of use. Cough, if not too troublesome, is best left alone. If it be dry or harassing, opium or codeine may be given at night to secure sleep. Hydrocyanic acid, belladonna, and the expectorants, in combination with codeine, often relieve this troublesome symptom, but care should be taken that the stomach be not disturbed by nauseant remedies. If the cough be accompanied with profuse expectoration, the expectorants are not indicated, but reliance should be placed upon creosote, turpentine and its derivatives, and the mineral acids.

Pain in the chest is to be treated by counter-irritation.

For the treatment of hæmoptysis see Pulmonary Hemorrhage.

Diarrhœa should be treated on general principles, but opium in some form has almost always to be used.

4. DISEASES OF THE PLEURA.

FIBRINOUS OR DRY PLEURISY; PLASTIC PLEURISY.

Etiology.—This form of pleurisy may be primary or secondary. The primary form may appear to be due to exposure to wet and cold, but modern theories regard cold merely as a predisposing factor to bacterial infection. The secondary form complicates any acute or chronic pulmonary disease involving the periphery of the lung. Thus, pleurisy occurs in conjunction with pneumonia with abscess, gangrene, or cancer of the lung, and with hemorrhagic infarctions. Occurring with pulmonary tuberculosis and phthisis, it may be the earliest indication of tubercular disease, and many cases of so-called "primary pleurisy" owe their origin to a small undiscovered tubercular lesion in the lung that may finally develop and give rise to symptoms.

Pleurisy may be secondary to inflammation of organs other than the lungs. Thus, pleurisy may arise from caries of the ribs or of the vertebræ, from perforation of an œsophageal cancer, from tubercular disease of the bronchial glands, from pericarditis, or from peritonitis. Cases following erysipelas of the chest-wall are not infrequent.

Patients with gout and with Bright's disease are more subject to pleurisy than are others. A pleurisy develops during the course of acute rheumatism, the pleura being involved, as are other fibro-serous membranes, as one of the regular manifestations of the disease. The belief is gaining ground that pleurisy, after all, results from the action of various micro-organisms, the most common of which are the streptococcus pyogenes, the pneumococcus, and the bacillus tuberculosis. Probably there are many other micro-organisms capable of causing the disease, and further bacterial examinations are desirable to enable cases of pleurisy to be grouped according to their microbic cause.

Pathology.—The pleura becomes congested, dry, and loses its normal lustre. Fibrin and serum infiltrate the thickness of the pleura and make their way to the free surface, so that the pleura is coated with a layer of lymph of variable thickness. The exudate may be shaggy in appearance or may be thick and stratified. Microscopically, the fibrinous exudate consists of fibrin, leucocytes, red blood-cells, and serum. The serum, however, is but slight in amount and undergoes rapid absorption. Subsequently the exudate becomes absorbed or becomes organized into connective tissue, so that the pleura is thickened and adherent to the opposing pleural surface.

Dry pleurisy usually begins in the pulmonary pleura and is limited to a small area. The opposing pleural surface usually is involved secondarily.

Symptoms.—The symptoms of secondary pleurisy are often masked by those of the primary disease. Pain in the side and the friction râle are the only characteristic symptoms.

In primary cases the attack may begin with a chill and with fever rarely over 102° F. The pain, which is sticking or stabbing in character, is referred to the site of the pleurisy. The pain is rendered worse by deep breathing or by coughing. There may be tenderness in the intercostal spaces over the lesion. The breathing is rapid and shallow, and there may be a dry, painful cough which is of reflex origin. In mild cases a stitch in the side on deep breathing may be the only symptom.

Physical Signs.—The characteristic physical sign of dry pleurisy is the pleural friction sound, which may be crepitant or subcrepitant or which may resemble a moist mucous râle. The crepitant râle is a fine dry crackle or shower of crackles heard at the end of inspiration only; it arises only in the pleura. A subcrepitant râle is a fine, moist, sticky sound, heard with inspiration, with expiration, or with both. Mucoid râles may arise from the rubbing together of surfaces covered with very moist lymph; they may exactly simulate the bronchial râles.

The differential diagnosis between pleural and bronchial râles is as follows:

Pleuritic Râles.	*Bronchial Râles.*
1. May be of the crepitant variety.	1. Never the crepitant variety.
2. Sound superficial, directly under the ear.	2. Sound "deep in."
3. Fairly constant.	3. Very inconstant.
4. Not influenced by coughing.	4. Influenced by coughing.
5. Over local area, which does not alter its position.	5. May be over large areas or in shifting areas.
6. All of one variety.	6. Usually assorted râles of all kinds.

The occurrence and diagnosis of the pleuro-pericardial friction sound has been described under Pericarditis (p. 187).

The absence of pleuritic râles does not necessarily exclude pleurisy, as the râles may come and go, may only appear on deep breathing, and may, moreover, arise at areas which are deeply seated, as in diaphragmatic or mediastinal pleurisy.

The **duration** of the disease is from three to ten days.

The **prognosis** of the attack itself is good, but a broader view must be taken than that of mere temporary recovery. The cause and the significance of the pleurisy and the sequelæ that may result from a thickened and adherent pleura must be considered.

Treatment.—During the attack the patient should be kept quiet, but need not necessarily be confined to the bed, or even to the house, unless the symptoms be severe. Counter-irritation by cupping, by iodine, or by blisters often diminishes the pain and checks the spread of the inflammation. Hot poultices are not so efficient as ice-bags applied locally. In all cases a brisk purgative should be given at the start, preferably calomel or magnesium sulphate. For the pain morphine may be given, and the chest may be strapped with adhesive plaster as for fractured rib, to diminish the friction between the inflamed pleural surfaces. In rheumatic cases salicylic acid or its derivatives should be given in full doses, as for acute articular rheumatism.

PLEURISY WITH EFFUSION.

Etiology and Synonym.—The etiology of sero-fibrinous pleurisy is the same as that of the plastic form. The former seems, however, to be due to a severer form of bacterial infection. Pulmonary tubercular disease follows, in time, one-third of the so-called "primary" cases. *Synonym:* Sero-fibrinous pleurisy.

The **pathology** of the sero-fibrinous is the same as that of the plastic form, except that there is added an excessive exudation of serum; moreover, the inflammation involves a larger area than in dry pleurisy. The exuded fluid is of a composition resembling that of blood-serum; its color is citron-yellow, and it may be clear, or somewhat turbid from flocculi of fibrin or from leucocytes and desquamated cells from the pleural surface. Blood may be present from rupture of fine blood-vessels or in the case of cachectic and debilitated subjects. The amount of the exudation varies greatly. An amount under 300 cubic centimeters does not give rise to physical signs in an adult. From one to two pints is the usual quantity, but eight to ten pints may be exuded. The exudation sinks to the dependent portions of the pleural sac unless encapsulated by previously existing adhesions—a somewhat rare occurrence in sero-fibrinous pleurisy. The fluid in the pleural sac rarely changes its level with any change in the position of the patient, being practically encapsulated by fibrinous adhesions between the lung above and the costal pleura. The upper level does not follow the ordinary laws of water-level, but follows a curve to which the name of "Garland's S-curve" is given.

Mechanical Effects of the Effusion.—The lung floats upward, its base resting on the fluid. As the fluid takes the place of the lung in the pleural vacuum, the lung is free to shrink, from its own elastic retraction, until the pleural sac is two-thirds full of fluid; when this point is reached the lung is in a condition of elastic equilibrium. Any excess of fluid over this amount exerts a direct pressure on the lung, so that in extensive effusions the lung is compressed, forming a dense, airless, carnified mass at the dome of the pleural cavity. The heart is bodily displaced to the opposite side, but it undergoes no twisting upon its axis, so that kinks in the great vessels do not occur. The diaphragm is sagged downward, and in right-sided pleurisy the liver is depressed. The intercostal spaces bulge, especially in children, and the affected side measures from one-half to one inch more than the other side.

The **symptoms** are *inflammatory* and *mechanical.*

1. *Inflammatory symptoms* may occur acutely or subacutely. If the onset be sudden, there may be a chill, which, however, is never so severe as in pneumonia. The

temperature rises to from 101° to 103° F., attains its maximum on about the third day, and slowly subsides, reaching the normal in from seven to ten days. The temperature is fairly continuous, not remittent as in empyema, and there is no definite crisis. Persistence of the fever after two weeks, or a temperature higher than 104° F. at any time, suggests empyema or tuberculosis. Prostration is in proportion to the severity of the inflammation and the fever. The pulse is rapid and compressible. Pain is marked at the onset, but it becomes less marked as the effusion is poured out, separating the opposing inflamed pleural surfaces. There may be a reflex cough with a scanty mucous expectoration. If the onset be insidious, the inflammatory symptoms are less marked. The chill is absent, the fever is rarely over 101° or 102° F., and prostration is so slight that the patient is up and frequently is able to work.

2. *Mechanical symptoms* depend upon the amount of the effusion and upon the rapidity with which it is poured out.

Dyspnœa results from the pleuritic pain and from the diminished expansion of the lung. It may be present only on exertion, or it may be so extreme as to be most distressing. The more rapidly the effusion is poured out, the more marked is the dyspnœa.

The position of the patient in bed is often suggestive. Before the effusion is poured out he lies upon the sound side, so as not to press the inflamed pleural surfaces together by his weight. After the effusion occurs he lies upon the affected side, so that the weight of the fluid will not embarrass the action of the heart or of the sound lung.

Cyanosis is likely to occur in cases with large effusions, and the heart's action may be weak and irregular.

The **physical signs** may be described as occurring before effusion, during the effusion, and after absorption of the effusion.

Before the effusion the friction râles of dry pleurisy are present, expansion is limited, and the percussion-note may be slightly dull.

During the Effusion.—There is an important distinction to be made between the physical signs of moderate and those of excessive exudation.

1. *The signs of moderate effusion* begin to appear when the exudation reaches ten or twelve ounces in adults or three or four ounces in children.

(*a*) *Below the level of the fluid* there should be diminished expansion, slight bulging of the intercostal spaces, especially in children, and an increased girth of the affected side. The percussion-note is flat, the upper limit of flatness describing "Garland's S-curve." This curve begins low in the back, rises to its highest point in the axilla, and then sinks with a slight descent to the sternum. The upper line of flatness is rarely influenced by a changed position of the patient. In left-sided pleurisy flatness replaces the normal tympany of Traube's semilunar space. Below the line of the fluid the voice and the breathing are muffled and even lost, and vocal fremitus should be absent. It is claimed that the whispered voice may be transmitted through serous, but not through purulent, effusions (Baccelli's sign).

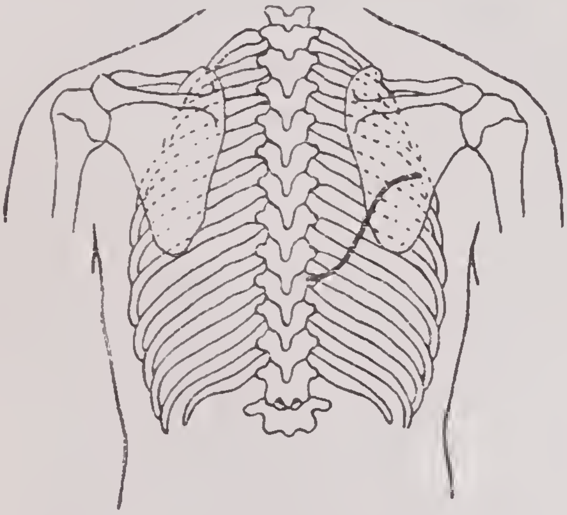


FIG. 41.—Garland's S-curve.

Exceptionally, below the level, voice and breathing may persist, though distant and indistinct; pleuritic râles may be heard through adhesions persisting below the level. In children soft bronchial voice and breathing may be heard, even if there be no compression of the lung. In some cases, especially in aged subjects, the percussion-note may be dull or dull-tympanitic below the fluid. Vocal fremitus may persist below the level, from adhesions extending downward through the effusion, or the fremitus may be transmitted along the chest-wall from the lung above or from the opposite side.

(*b*) *At the level of the fluid* there should be dulness and pleuritic râles. Exceptionally, a bleating of the voice (œgophony) is heard, being elicited by having the patient pronounce the words "want" or "plant." Œgophony, however, is a sign of rare occurrence. Pleural râles may be absent at the line of fluid.

(*c*) *Above the level of the fluid*, in moderate effusions, the physical signs may be normal, or there may be a tympanitic percussion-note with feeble breathing. Tympany in these cases is due to relaxation of the lung-tissue; it is most marked under the clavicle, constituting "Skoda's sign." In some cases in children the note under the clavicle may even be of the "cracked-pot" order, and may lead to the erroneous diagnosis of a cavity, especially as cavernous breathing may be heard, by reason of a large bronchus approaching the chest-wall owing to the retraction of the lung. Both bronchial and pleural râles are often heard over the retracted lung.

(*d*) *Signs of Displaced Viscera.*—In left-sided pleurisy the apex beat of the heart may be under the sternum, so that it cannot be appreciated, or in more abundant effusion it may be displaced as far as the right nipple. In right-sided pleurisy the apex beat may be as far to the left as the mid-axilla. Over the apex of such a displaced heart a systolic murmur may be heard. In right-sided pleurisy the lower border of the liver is felt by palpation below the free border of the ribs in the mammary line.

2. *The signs of excessive exudation* differ from the preced-

ing physical signs in that the lung begins to be actually compressed. Over the compressed lung the note is dull-tympanitic or dull, the breathing and the voice become broncho-vesicular or bronchial, and vocal fremitus is increased. There may be loud bronchial râles of a gurgling quality. As the lung begins to be compressed "Garland's S-curve" becomes less marked until finally the upper limit of the flatness is a straight horizontal line. Below the level of the fluid the percussion-note is flat, and breathing and voice are of a soft, distant, bronchial character, being transmitted down the tense chest-wall from the compressed lung above. In the same manner vocal fremitus may be transmitted down the chest-wall over the fluid, although, as a rule, vocal fremitus is absent in these cases. The chest is usually motionless on inspiration, the intercostal spaces are bulged or tense, mensuration shows an increased growth of from one to one and a half inches, and the displacement of the heart and the liver becomes evident.

Patients with the above physical signs are often treated for pneumonia, but a mistake in diagnosis should not occur, for, if doubt exist, the aspirating-needle should be used.

As the effusion becomes absorbed the physical signs of fluid disappear and breathing is heard to the base of the chest. The friction râle usually reappears. Some dulness on percussion and feeble breathing with friction râles persist for months, from thickening of the pleura.

In every case of doubt an aspirating-needle should be inserted, with the strictest antiseptic precautions, below the supposed level of the fluid. Fluid may not appear at the first introduction of the needle, should its calibre be blocked by a bit of fibrin. The use of the aspirating-needle not only makes positive the diagnosis of fluid, but it determines the character of the fluid—whether serous, hemorrhagic, or purulent.

Course of Pleurisy with Effusion.—1. The onset may be acute, both inflammatory and mechanical symptoms being pronounced. Inflammatory symptoms subside in from seven to ten days; later the effusion becomes absorbed or is removed, mechanical symptoms disappear, and the patient recovers.

2. In other cases the inflammatory symptoms subside, but the fluid remains unabsorbed and mechanical symptoms persist.

3. Other cases begin as pleurisy with effusion, but gradually septic symptoms develop. The fever becomes high and remittent; there are erratic chills and night-sweats with rapid emaciation. The aspirating-needle shows the fluid to have become purulent from an added infection by pus cocci. These cases, however, are rare.

4. The onset may be insidious. Inflammatory symptoms are not marked, but mechanical symptoms gradually increase. These patients are the ones who feel "run down" and short of breath on exertion, and who come to the hospital with a chest half full of fluid.

5. There may be a double pleurisy. These cases are usually of tubercular origin. Their course is persistent and insidious, and pericarditis is a frequent complication.

Sudden death is a rare termination, being more common with the severer cases of pleurisy. Post-mortem examination shows in some cases congestion and œdema of the lungs, and in others an ante-mortem heart-clot, while in other cases no pathological cause can be found.

Sequelæ.—In some cases the pleura returns to a practically normal condition. In others the pleura is left thickened and adherent and may lead to the following complaints:

1. Local tenderness, worse on exertion or in damp weather.

2. Reflex cough.
3. Pain and slight dyspnœa on exertion.
4. There may be developed in the course of time—(*a*) chronic bronchitis; (*b*) emphysema; (*c*) interstitial pneumonia; (*a*) chronic pleurisy, with the formation of new connective tissue; or (*e*) recurring attacks of pleurisy with fibrin or with effusion.

The heart may remain fixed in its abnormal position by adhesions, or it may ultimately be displaced to the affected side by retraction of pleuritic adhesions.

The **prognosis** for the attack itself is good. Neglected cases in which the lung has been compressed may, however, do badly. The ultimate prognosis should consider the underlying cause of the pleurisy and the possibility of sequelæ arising from pleural thickening and from adhesions.

Treatment.—During the inflammatory stage the patient should be put to bed and be kept on a light diet. Pain should be treated by counter-irritation, strapping, hot or cold applications to the chest, and, if necessary, by opium by the mouth or subcutaneously. Painful cough is to be relieved by sedatives. In rheumatic cases salicylates, with or without potassium iodide, may be given.

The treatment of the mechanical symptoms is designed to get rid of the effusion, and a choice of two methods presents itself:

The reduction of the effusion may be accomplished by free purgation and diuresis. The best cathartic for the purpose is magnesium sulphate in doses of from 1 to 1½ ounces, given every second morning, an hour before breakfast, in a concentrated form. The choice of the proper diuretic is simply empirical: one after another should be tried, singly or in combination, until the desired effect is accomplished. The diet should be concentrated, and only a minimum quantity of water should be allowed.

Withdrawal of the effusion by aspiration is the most satisfactory treatment, and it should be resorted to under the following conditions: (1) A sudden large effusion with dyspnœa and cyanosis. (2) A large effusion with marked mechanical symptoms. (3) Should the physical signs of compression of the lung be found. Aspiration in these cases should be done without delay. (4) Should the effusion be uninfluenced by catharsis and diuretics. Absorption proceeds better, even if but little fluid be withdrawn. It certainly seems wrong to waste much time and debilitate the patient with exhausting treatment when so safe and efficient a means of relief may be used.

To aspirate, the patient should be semi-recumbent. Stimulants should be at hand, and the strictest asepsis should be employed. The needle is inserted below the level of the fluid, usually in the eighth space in the axillary line, and the fluid is withdrawn slowly. No more than 50 ounces should be withdrawn at any one time, and the operation should be stopped at once if severe pain, dyspnœa, faintness, or paroxysmal cough develop. The danger of aspiration is sudden heart failure, but this is exceedingly rare. After aspiration the remaining fluid is usually absorbed, but occasionally the fluid reaccumulates, and it must be removed by a second or even a third aspiration.

PURULENT PLEURISY (EMPYEMA).

Etiology.—Purulent pleurisy is regularly due to the infection of the pleural cavity by some micro-organism capable of exciting suppuration. Infection is due to the following bacteria in order of frequency: Streptococci, pneumococcus, tubercle bacilli, staphylococci, Eberth's bacilli, and the saprophytic bacteria of gangrene. These micro-organisms may infect in pure cultures or in combinations (mixed infection).

Infection is permitted by—(1) Penetrating wounds of the chest-wall, or by the use of a septic aspirating-needle; (2) gangrene, abscess, or septic emboli of the lung, ruptured tubercular cavities, or perforation of the œsophagus; (3) infection through the diaphragm following abscess of the liver or peritonitis; (4) secondary to pneumonia and to some acute infectious diseases, as scarlet fever, typhoid, measles, whooping-cough, and "grippe."

Pathology.—The lesion of purulent pleurisy is the same as that of pleurisy with serous effusion, except that pus-cells infiltrate the thickness of the pleura and are found in the effusion in varying amounts. The exudate may be sero-pus, or thick and creamy, or of a greenish or yellowish color. The odor is usually mawkish, but it may be foul or gangrenous. A peculiar yeasty odor is noticed in many cases of pneumococcus infection. The effusion is more apt to be sacculated than that of serous exudation, and compression of the lung is more apt to occur. Subsequent changes may occur in the pleura, in the effusion, and in other viscera.

Changes in the Pleura.—1. In rare cases, especially in children and in pneumococcus infection, with prompt removal of the exudate the pleura may return to a healthy condition.

2. The pleura may become thickened and adherent after the removal of the fluid.

3. The pleura may be thickened; its surface is composed of granulation-tissue secreting pus, constituting a pyogenic membrane.

4. In the pleura may be deposited the salts of lime.

5. The pleura may become necrotic in places and slough. By the extension of the necrotic processes the pus may find its way through the chest-wall (empyema necessitatis), or through the diaphragm, or it may rupture into the lung or the pericardium.

Changes in the effusion result by an added infection of putrefactive germs, usually those of gangrene of the lung. The effusion becomes foul and offensive, and gases of decomposition may form, constituting pyo-pneumothorax. Small purulent effusions may become inspissated and infiltrated with lime-salts.

Changes in other viscera are those changes common to prolonged suppuration. There may be amyloid degeneration of the spleen, the liver, and the kidneys, or chronic diffuse nephritis.

Symptoms of inflammatory and mechanical origin are present, resembling those of fibrino-serous pleurisy, but in empyema septic symptoms are added, consisting of erratic chills, high remittent temperature, cold sweats, prostration, diarrhœa, emaciation, and the development of a septicæmic or typhoid condition.

1. The onset may be sudden, with inflammatory and mechanical symptoms. There are a chill, fever, pain in the side, prostration, dyspnœa, and the physical signs of pleural effusion. The case resembles at first fibrino-serous pleurisy, but the aspirating-needle draws pus. Later, septic symptoms develop.

2. The onset may be insidious. Inflammatory symptoms, such as initial chill and fever, are not marked, but mechanical and septic symptoms slowly develop. The patient is indisposed and has pain in the side, slight fever, and dyspnœa. The fever becomes higher and is remittent. Erratic chills, cold sweating, and prostration become marked.

3. If empyema follow pneumonia, there is usually an attempt at crisis. The temperature, however, rises again and becomes remittent, dyspnœa develops, septic symptoms appear, and the physical signs of a pleural effusion make their appearance. Chills, however, are not common in pure pneumococcus infection of the pleura.

Course of the Disease.—1. Some patients die during the acute onset, from the intensity of the inflammation.

2. Septicæmia may be developed in early cases, especially if the effusion undergoes putrefactive changes.

3. Some patients pass into a hectic condition and die in several months, exhausted or in the typhoid condition.

4. Death may result from the primary diseases, as phthisis, gangrene, or abscess of the lung.

5. Death may result from perforation into the pericardium or the peritoneum. If rupture into a bronchus occurs, pus will be expectorated, with a relief of all the symptoms. Owing to poor drainage, the improvement is usually temporary. In rare cases, however, spontaneous cure has been effected.

Aspiration of the pus into the bronchi at the time of rupture may cause asphyxia or septic broncho-pneumonia, and pneumothorax may develop by air entering the pleural cavity through the bronchial fistula.

If rupture through the chest-wall occurs, the opening is usually in the fifth or sixth interspace in front. Drainage is usually poor, and improvement is but temporary. Spontaneous cure may, however, result, with or without the formation of a thoracic fistula.

6. Small circumscribed empyemata may terminate by gradual absorption, by thickening and calcification of the pleura, and by local chest-retraction.

The **physical signs** are in the main those of fibrino-serous pleurisy. Mention, however, should be made of a few additional points:

The interspaces are apt to be obliterated more than in simple pleurisy, and they may even bulge. There may be œdema of the chest-wall. Whispered speech is not usually transmitted through purulent effusions.

In children distinct bronchial breathing may be heard over a large purulent effusion, so that a mistaken diagnosis of pneumonia frequently is made.

Pulsations of the effusion synchronous with the cardiac systole (pulsating empyema) is sometimes observed, for which no satisfactory explanation can be offered. Of 42 cases, 39 occurred on the left side.

Prognosis.—Empyema is a very serious affection, the severity of which largely depends upon the particular micro-organism to which it is due. Infection by the pneumococcus is regularly less severe than that caused by the streptococci. Cases due to the saprophytic bacteria of gangrene afford the worst prognosis. The prognosis is better in children than in adults, in cases promptly treated than in those allowed to progress, and in cases with slight sepsis. The prognosis also depends upon the general condition of the patient and the nature of the primary disease.

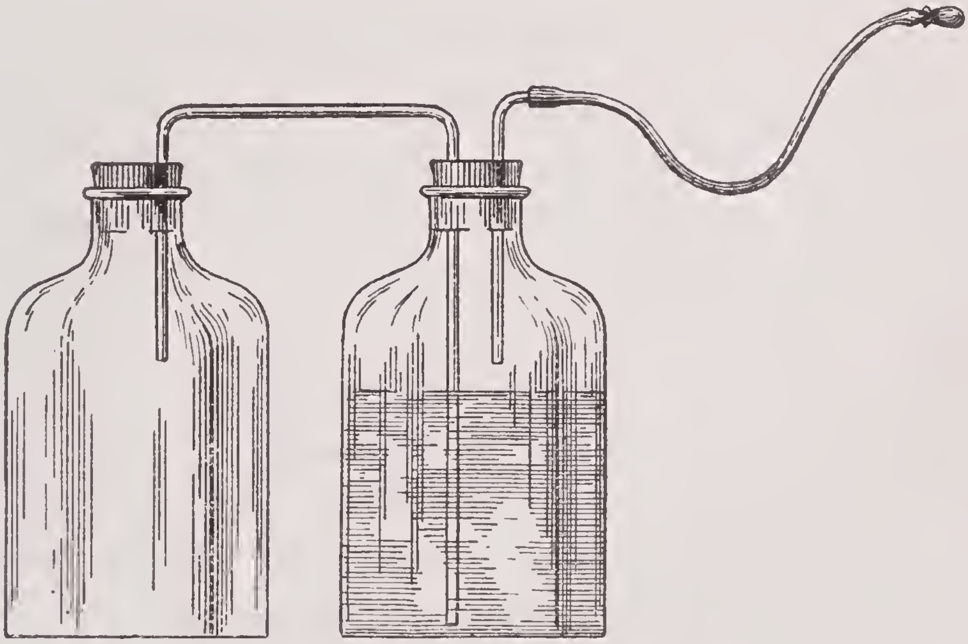


FIG. 42.—Arrangement of bottles for forced expiration.

The **treatment** of empyema is that of an abscess requiring incision and drainage. Cases due to simple infection by pneumonia, especially in infants, may recover after aspiration, but, as a general rule to which there are but few exceptions, aspiration should not be resorted to except as a temporary measure. Incision and drainage should be insisted upon, no matter how desperate the patient's condition may seem. The details of the operation are to be found in text-books on surgery. Irrigation is permissible only with putrid or gangrenous empyema. Chest-gymnastics calculated to induce deep inspiratory efforts are of value in convalescence. The method of Ralston James, of forced expiratory efforts, is to be advised to expand the retracted lung. By the arrangement of the water-bottles shown in Figure 42, water may be forced from one bottle into the other by blowing into the mouth-piece. This process should be repeated a number of times daily.

Should the lung not expand, and should a thoracic fistula result from the operation, Estländer's operation may be performed. This operation consists in the resection of one or more inches of several ribs in the lateral aspect of the chest.

CHRONIC PLEURISY.

Etiology and Synonyms.—A thickened and adherent condition of the pleura may result from acute pleurisy, causing, in some cases, diminished chest-expansion, pain aggravated by deep breathing or by cold and damp weather, and possibly a reflex cough. Aside from these symptoms, cases are met with in which the pleura is the seat of a progressive and chronic inflammation, to which the name "chronic pleurisy" should more properly be applied. Chronic pleurisy may follow acute pleurisy, whether plastic, fibrinoserous, or purulent, or the inflammation may be chronic from the start. *Synonyms:* Chronic adhesive pleurisy; Pleurisy with the production of new connective tissue.

Pathology.—Two forms of the disease are described:

1. *Chronic Dry Pleurisy.*—The lesion is usually confined to one pleura, but it may be bilateral. The pleura is thickened by growth of connective tissue, so that it may exceed

one-half or even three-fourths of an inch in thickness, and the opposing pleural surfaces are adherent. This form of pleurisy occurs most extensively after empyema, is one of the regular lesions of chronic pulmonary phthisis, and in rare cases may appear at the base as a primary disease.

2. *Chronic Pleurisy with Effusion.*—The pleura is thickened; there is a serous effusion which in many cases is sacculated, the encapsulated serum being encysted by thickened and adherent pleura. In cases following empyema there may be collections of inspissated pus containing lime-salts. The lung, which may be invested with thickened pleura preventing its full expansion, may be the seat of a bronchitis or of a chronic interstitial pneumonia with or without bronchiectasis. The heart may be displaced to either side.

The **symptoms** at first are slight. There is a pleuritic pain, worse on deep breathing and in damp weather, freqently associated with tenderness in the overlying intercostal spaces. Dyspnœa on exertion results from diminished chest-expansion; it is more marked in cases with serous effusion. There may be a cough of reflex origin or due to a complicating bronchitis. The patient loses flesh and strength and becomes anæmic. These symptoms become more and more marked as the disease progresses.

Physical Signs.—Inspection shows retraction of the chest-wall and diminished expansion. If the pleurisy be dry, there will be dulness or flatness according to the thickness of the pleura and the strength of the percussion-blow. Breathing and voice-sounds are muffled, distant, or absent altogether. Vocal fremitus is diminished or lost, but pleuritic fremitus may be marked on forced inspiration. Auscultation reveals pleuritic râles, creakings, and old rubbing friction sounds.

If there be fluid, the physical signs of pleural effusion will be present, although the signs are often obscured by those of the surrounding thickened pleura, especially if the effusion be encapsulated. There may be added the physical signs of bronchitis, bronchiectatic cavities, and interstitial pneumonia.

The **diagnosis** of thickened pleura from fluid is to be made with certainty only by the use of the aspirating-needle. If the pleura be thickened, the needle is felt to pass through a firm, almost cartilaginous substance which gives a characteristic feeling of resistance. The diagnosis of chronic pleurisy from new growths of the pleura often presents great difficulties.

The **course** of the disease is slow and steady, extending over years. The patient is finally reduced to invalidism, but death almost always results from some intercurrent disease.

Treatment consists (1) in building up the general health by good food, proper climate, and tonic medication; (2) in counter-irritation by iodine or by blisters; (3) by exercises destined to increase chest-expansion; and (4) by the removal of serous accumulations in small quantities at a time.

TUBERCULAR PLEURISY.

Etiology.—The association of pleurisy, both plastic and with effusion, and pulmonary tuberculosis has elsewhere been described. Besides these cases the pleura may be the seat of a tubercular disease, either as one of the lesions of acute general miliary tuberculosis or as a localized lesion which may be primary or secondary to other tubercular deposits, especially in the bronchial glands.

Pathology.—The lesions are usually confined to one side, but in rare instances they may be bilateral.

1. The pleura is thickened with tubercular nodules, cheesy masses, and leucocytes, and miliary tubercles appear upon the free surface. The appearance of the pleura and the character of the effusion vary in different cases.

(*a*) The pleura may be deeply congested and be studded with miliary tubercles. There is a hemorrhagic serous exudation.

(*b*) The pleura is studded with tubercles and covered by fibrin. There is a fibrino-serous effusion.

(*c*) The pleura is thickened with caseous and softening tubercular nodules, its surface is coated with fibrin, pus, and cheesy matter, and it may present tubercular ulcerations. The effusion is purulent and contains little cheesy masses and shreds of the pleural tissue. It is remarkable that in a large proportion of these cases neither tubercle bacilli nor the germs of suppuration are found in the effusion; but inoculation of this apparently sterile fluid in animals is frequently followed by the development of tubercles.

2. There may be a tubercular dry pleurisy thus described by Osler: "Both parietal and costal layers are greatly thickened—perhaps from two to three millimeters each—and present firm fibroid caseous masses and small tubercles, while uniting these two greatly thickened layers is a reddish-gray fibroid tissue, sometimes infiltrated with serum. This may be a local process confined to one pleura, or it may be in both." These cases are frequently combined with a similar condition of the pericardium and the peritoneum.

The **symptoms** of tubercular pleurisy resemble in character those of the non-tubercular forms, but run a slow, insidious course. Acute cases are rare.

Diagnosis is made by attention to the following particulars: (1) There is usually some antecedent tubercular history, or evidences of a pre-existing tubercular disease. (2) The onset of the pleurisy is insidious. (3) The course of the disease is progressively bad. (4) The fluid does not disappear with medication: it persistently reaccumulates after aspiration. (5) Emaciation and prostration are out of proportion to the local disease. (6) Aspiration shows a purulent effusion containing cheesy matter in which tubercle bacilli may be found, or the serum may be hemorrhagic. If cancer of the pleura and laceration of the lung by the point of the needle be excluded, the occurrence of a hemorrhagic serum is almost diagnostic of tubercular pleurisy.

Duration.—Rare acute cases may terminate in two weeks. The usual duration is from three to six months or even longer.

The **prognosis** is regularly fatal.

Treatment consists in building up the general health by the rules laid down for the treatment of tuberculosis, and in the withdrawal of the fluid by aspiration when it accumulates. Should the effusion become purulent, the case should be treated as one of empyema—by incision and drainage.

PNEUMOTHORAX; HYDRO-PNEUMOTHORAX; PYO-PNEUMOTHORAX.

Etiology.—Pneumothorax arises—(1) From perforation of the chest-wall following penetrating wounds, incision for the drainage of empyema, or empyema necessitatis. (2) From the perforation of the pulmonary pleura by rupture of the lung by violence, by overstraining, or by injury from careless aspiration; or there may be rupture of emphysematous vesicles. Air may dissect down the peritracheal connective tissue, from violent coughing attacks following tracheotomy whenever the tube becomes blocked, and may rupture into the pleural cavity. One fatal case of the writer's occurred in this way. Rupture of a tubercular cavity is the direct cause of 90 per cent. of all cases of pneumothorax. Less common are the cases due to septic broncho-pneumonia, abscess or gangrene of the lung, hemorrhagic and septic infarcts, and rupture of an empyema into a bronchus. (3) From the perforation of other organs, as from cancer of the œsophagus, the stomach, or the colon. The accident may follow perforation through the diaphragm of a subphrenic pyo-pneumothorax.

Pathology.—When air enters, the pleural vacuum is at once destroyed, and the lung shrinks by reason of its own elasticity, the heart is displaced bodily toward the opposite side, and the liver sags downward, exceeding the downward displacement observed in pleural effusion. If the point of perforation remain open, the intra-pleural air is at atmospheric pressure, and the lung is not compressed. If the orifice of rupture be valve-like (ventilating pneumothorax), air can enter during inspiration, but its exit during expiration is prevented, so that the intra-pleural pressure becomes raised and the lung becomes compressed and carnified. The point of rupture may be large, constituting a pleurobronchial fistula, especially in long-continued cases, or it may be small, baffling detection on post-mortem examination. If the orifice of rupture be closed by a contraction of the lung or by a deposit of fibrin, the intra-pleural air may be at any degree of tension. The question of tension is of importance in the understanding of the physical signs.

In rare cases the entering air is sterile, and a simple pneumothorax results; usually, however, infection and inflammation of the pleura result in the formation of a serous effusion (hydro-pneumothorax) or of pus (pyo-pneumothorax), the latter condition being far the more common. The effusion sinks to the dependent portion of the pleural cavity; its upper level is a straight horizontal line (there being no Garland's S-curve, as with pleurisy with effusion), and the level of the fluid changes regularly with the varying position of the patient. In rare instances the orifice of perforation is surrounded by pleural adhesions, so that a localized sacculated pyo-pneumothorax results.

Symptoms.—The symptoms may be sudden and urgent or may be latent or obscure.

1. If pneumothorax occur in a fairly healthy patient, the onset is sudden and alarming. There is a severe pain in the side, with a feeling that "something has given way." There is extreme dyspnœa, amounting to agonizing air-hunger, with cyanosis in some cases, and aphonia "for want of breath" is usually observed. Symptoms of surgical shock rapidly develop—lividity, prostration, cold, clammy skin, feeble and rapid heart-action—and death may result from shock within a few hours. Should the patient survive, the symptoms of shock slowly disappear. Dyspnœa continues, with rapid, insufficient breathing. The patient sits up with the body inclined to the affected side. There are evidences of poor circulation—lividity, dropsy, or venous congestions. Pain in the side continues, and the symptoms of pleural effusion make their appearance. Death finally results from exhaustion, from sepsis, or from pre-existing disease.

2. If pneumothorax occur in a person much debilitated by phthisis or by pulmonary gangrene, the symptoms are obscured. Increased dyspnœa and enfeebled heart-action may be the only additional symptoms. In some cases sudden death results. In rare cases, in healthy adults with pneumothorax, the disease runs this insidious and obscure course.

3. If the pneumothorax be due to rupture of pulmonary air-vessels after severe straining efforts, the air is usually absorbed, but inflammation of the pleura with serous effusion almost regularly results.

Physical Signs.—On inspection the affected side is enlarged and motionless. Vocal fremitus is diminished or lost. The percussion-note depends upon the tension of the intrapleural air. If the air be at low tension, the note will be tympanitic or amphoric. If there be a free pleuro-bronchial fistula, a "cracked-pot" note may be obtained. If the intra-pleural air be at high tension, the percussion-note will be dull-tympantic, dull, or even flat. Percussion over a pneumothorax frequently gives the sensation of percussing an air-cushion. Flatness is obtained over the effusion; the upper limit of the flatness is horizontal and changes according to the position of the patient.

Breathing- and voice-sounds may be feeble and distant, contrasting with the exaggerated breathing-sounds over the sound side; or there may be a distant inspiratory murmur of amphoric quality. When the orifice of rupture admits air freely, voice and breathing may be typically amphoric.

Should the lung be compressed, there will be bronchial voice and breathing over the carnified lung, heard with less distinctness over the air. The Hippocratic succussion consists of loud splashing sounds heard when the patient is violently shaken. Similar sounds may occur, however, when gas and fluid are shaken in a distended stomach. The metallic tinkle consists of clear tinkling sounds resembling those produced by striking a pin against a thin glass tumbler. The "penny-click" of Trousseau is one of the most characteristic physical signs of pneumothorax. A coin pressed firmly in an intercostal space in front is tapped with another coin while the auscultator listens at the back of the chest: the transmission of a metallic echoing sound is characteristic of a large air-cavity; it is not, however, pathognomonic of pneumothorax.

Aid is afforded in diagnosis by the physical signs of displaced liver and heart.

The **diagnosis** is usually easy. Mistakes may arise by confusing pneumothorax with—(*a*) Large phthisical cavities at the base of the lung. Here the penny-click is not heard, succussion is rarely obtained, pleuritic râles are heard generally over the area, and the heart and the liver are not displaced. There are, moreover, no sudden urgent symptoms.

(*b*) Diaphragmatic hernia following crush or injury. (*c*) Subphrenic pyo-pneumothorax.

Prognosis.—But few cases of pneumothorax recover. The prognosis depends upon the cause, the septic character of the infection of the pleura, the general condition of the patient, the presence of pre-existing disease, and the severity of the shock and the reaction.

Duration.—The ordinary duration of the disease is between two and three months. Patients may die in shock in a few hours, while in rare cases the disease becomes chronic and extends over months or years.

Treatment.—At the time of the perforation morphine or chloroform may be given for the pain, and the symptoms of shock may be treated on general principles. Where effusion forms, the case should be dealt with as ordinary pleurisy with effusion or as empyema—by aspiration, or incision and drainage. In pyo-pneumothorax occurring in advanced phthisis it may be the better course simply to remove the pus by aspiration rather than to render the last days of the patient uncomfortable by a surgical operation.

NEW GROWTHS OF THE PLEURA.

The majority of new growths in the pleura are of secondary nature and complicate tumors of the lung or the chest-wall, the pleura being directly invaded by the new growth. Of the primary new growths of the pleura, endothelial carcinoma is the most important. The pleura is infiltrated and studded with scattered nodules, or the cancer may be diffuse. Pleurisy is developed with fibrino-serous effusion, which in 12 per cent. of all cases is of a hemorrhagic character. Secondary metastatic growths may occur in the lungs, in the bronchial glands, or in distant organs.

The **symptoms** resemble those of chronic pleurisy with effusion. Pain, however, is more continuous and severe, and there may be exquisite tenderness in the intercostal spaces. The effusion may be hemorrhagic, and cancerous cachexia develops.

Diagnosis.—Aid in diagnosis may be afforded by the presence of malignant tumors elsewhere. The diagnosis in the earlier stages may with difficulty be made from chronic tubercular pleurisy.

The **physical signs** are those of thickened pleura with effusion.

The **prognosis** is invariably fatal.

The **treatment** is simply symptomatic. If sarcoma of the pleura be suspected, hypodermic injection of the toxic products of erysipelas germs may be employed.

HYDROTHORAX.

Etiology.—Hydrothorax occurs with dropsy of other organs, with nephritis, with diseases of the heart, and with profound anæmia; it may result from pressure of a tumor upon an intrathoracic vein.

Pathology.—The lesion of hydrothorax consists of an accumulation of serum in the pleural cavity, without inflammation of the pleura itself. In the cases due to nephritis or to anæmia the lesion is usually bilateral. In heart disease one cavity alone is involved in the majority of instances; if both cavities are implicated, however, the amount of the fluid in the two sides is not equal. Intrathoracic pressure on a vein regularly results in unilateral hydrothorax. The fluid is simple serum without inflammatory ingredients, the pleural surfaces are normal, and the amount of effusion is rarely excessive.

The **symptoms** are often obscured by those of the primary disease. Dyspnœa is, however, increased by the transudation, and it may be associated with cyanosis and great distress. Pain and fever are absent.

The **physical signs** are those of pleural effusion. The friction râle is not heard, however, and, as fibrinous adhesions do not exist, the fluid is more apt to change its level with the varying position of the patient than is common in fibrino-serous pleurisy. Compression of the lung rarely, if ever, occurs.

The **prognosis** is that of the primary disease.

The **treatment** also is that of the primary disease. The dropsies are to be treated by heart-stimulants, diuretics, and cathartics. If the breathing be embarrassed by the transudation, and no relief follows medicinal measures, repeated aspirations are to be resorted to.

HÆMOTHORAX.

Etiology.—Hemorrhage into the pleural cavity may occur from rupture of an aneurysm, from erosion of an intrathoracic vessel, or from injuries resulting in fractures of the rib or in laceration of the lung.

Pathology.—The blood may coagulate and be absorbed if not too excessive in amount. If infection occurs, there may be pleurisy with effusion or empyema.

The **symptoms** are those of hemorrhage—pallor, dyspnœa, thready pulse, sighing respirations, and restlessness, associated with pleuritic pain and dyspnœa. Symptoms of pleurisy with effusion or of empyema may develop in infected cases.

The **physical signs** are those of pleural effusion. The friction râle is, however, absent.

Prognosis.—A large hæmothorax, such as arises from rupture of an aneurysm, is rapidly fatal. Small hemorrhages, especially those due to injury, may terminate in absorption and recovery.

The **treatment** is that of acute anæmia—by transfusion, warmth to the extremities, and small doses of opium. A small hæmothorax is best left alone. If the clot be infected or be large enough to interfere with respiration, it may be evacuated by incision.

5. DISEASES OF THE MEDIASTINUM.

LYMPHADENITIS.

Simple lymphadenitis follows inflammations of the lungs or the bronchi, especially in children. More rarely the condition arises in the course of some infectious diseases, especially typhoid fever and diphtheria.

The *lesion* consists of swelling and congestion of the gland, resulting either in resolution or in enlargement. Suppuration rarely occurs. Not infrequently the glands become secondarily infected by the tubercle bacilli.

The adhesion of an enlarged gland to the œsophagus may result in a traction-diverticulum.

The *symptoms* are rarely observed. In some cases bronchitis with paroxysmal cough results from congestion and irritation of the neighboring structures.

Suppurative lymphadenitis may follow simple or tubercular inflammation of the glands. The pus may finally be inspissated, infiltrated with lime-salts, and encapsulated, or it may rupture into the bronchi or the œsophagus.

Tubercular lymphadenitis regularly accompanies tubercular lesions in the lung. In other cases the glands filtering out the impurities gaining entrance to the lungs may primarily be infected. The tubercular glands may attain a large size and may cause the pressure-symptoms of a mediastinal tumor. The caseous masses may become inspissated and encapsulated, or they may rupture into neighboring organs. The lungs, the pleura, or the pericardium may be involved secondarily by extension. General tuberculous infection so commonly results, especially in children, that search should be made for tubercular glands of the mediastinum in every case of acute miliary tuberculosis of obscure origin.

MEDIASTINAL TUMORS.

Of 520 cases of mediastinal tumor reported by Hare, cancer occurred in 134, sarcoma in 98, and lymphoma in 21. Less frequently are found dermoid and hydatid cysts, fibroma, lipoma, gumma, and enchondroma. Growths formed by the aggregation of tubercular glands and aneurysms have previously been described. Cancer may be primary or secondary. Sarcoma is more apt than cancer to be primary, men are more frequently affected than women, and the majority of cases occur between the twenty-fifth and fortieth years.

The **symptoms** are due to increasing intrathoracic pressure. Dyspnœa is the most marked symptom; it is due to pressure on the trachea, the recurrent laryngeal nerve, the lungs, or the bronchi. In the latter stages of the disease orthopnœa is usually developed. Cough may be paroxysmal, resembling that of whooping-cough, or there may be a brassy cough as with aneurysm. Pressure on the thoracic duct occasions rapid emaciation. Dysphagia occurs if the œsophagus be compressed. Compression of the thoracic veins results in cyanosis of the chest, the arms, the head, and the neck, and in extraordinary attempts to establish collateral circulation. Congestion and œdema of the lung may be occasioned by pressure on the pulmonary vein. Pleural effusion is apt to appear either by an involvement of the pleura by the new growth or from pressure of the tumor on the vena azygos or on one of the intercostal veins.

Pain is not as common with tumor as with aneurysm.

Physical Signs.—There is evident dyspnœa. Some valuable aid in localizing mediastinal new growths is afforded by noticing what position of the patient best relieves the pressure-symptoms and modifies the dyspnœa. There may be blueness of the upper part of the body and arms, while the enlarged thoracic and anastomosing abdominal veins stand forth prominent and distended. According to Osler, the distention and enlargement of the thoracic veins are more marked with lymphadenoma than with cancer or with sarcoma. The sternum or the intercostal spaces on either side may be bulged forward or may be involved by the growth. A transmitted expansion frequently suggests aneurysm, but it is not so marked, there is no lateral expansion, and there is no diastolic shock. The tracheal tug is seldom if ever obtained, and over the tumor no murmur is detected, as in the latter disease. Dulness is elicited by percussion over the growth, either over the upper sternum or between the spinal column and the scapula in case of involvement of the posterior mediastinum. The breathing on either side may be feeble from bronchial compression or may assume a tubular character. The physical signs are modified by the signs of pleural effusion or of cancer of the lung or the pericardium.

Diagnosis.—Many points of differential diagnosis from aneurysm of the aorta have already been alluded to. In many cases a positive diagnosis cannot be given, although, should the patient live over eighteen months, a malignant mediastinal growth may probably be excluded.

The **prognosis** depends upon the nature of the growth.

Treatment.—In cases of lymphoma the administration of arsenic is often followed by a diminution of the growth. For radical cure surgical treatment alone can avail, but as this is rarely possible, the medicinal treatment is merely palliative, to quiet the pain and to relieve the dyspnœa.

Opium may be given without conscientious dread of forming a habit.

ABSCESS OF THE MEDIASTINUM.

This affection is usually of traumatic origin; it may, however, be secondary to infectious fevers, to pyæmia, to erysipelas, or to suppurative disease of the adjacent viscera. Chronic abscesses are usually of tubercular origin. The abscess, which is usually situated in the anterior mediastinum, is more common in males than in females. The pus may finally become inspissated and encapsulated, or it may rupture through the sternum, through an intercostal space, or into the œsophagus or the trachea, or it may burrow into the abdominal cavity.

The **symptoms** are those of abscess and pressure. Pain is marked from the start, is of a throbbing character, and is associated with exquisite tenderness. Irregular fever, chills, and sweating mark the presence of pus. Cough, dysphagia, and dyspnœa occur as pressure-symptoms.

The **prognosis** must be guarded, owing to the possible complications.

Treatment.—In the earlier stages the ice-bag or the cold Leiter coil should be employed continuously. When pus has formed it may be evacuated by trephining the sternum.

EMPHYSEMA OF THE MEDIASTINUM.

This condition is met with in trauma, follows the operation of tracheotomy, and may result from rupture of the peripheral air-vesicles of the lung during violent coughing or straining. Air may enter the cellular tissue of the mediastinum by perforation of ulcers of the trachea, the bronchi, or the œsophagus. The emphysema may be limited to the mediastinum, may rupture into the pleura, causing pneumothorax, or may spread to the neck and even to the entire body. The *treatment* is entirely symptomatic.

MEDIASTINAL HÆMATOMA.

Hemorrhage into the mediastinal tissues occurs in hemorrhagic conditions, from erosion or rupture of blood-vessels or from rupture of an aneurysm.

The symptoms are those of hemorrhage and mediastinal pressure, while ecchymoses may appear after a few days in the lumbar region.

DISEASES OF THE THYMUS GLAND.

Hypertrophy is occasionally met with in children; it may cause spasm of the glottis (thymic asthma) or sudden death.

Abscess may develop in syphilitic children.

Sarcoma and **carcinoma** may originate in the thymus gland and may give the regular symptoms of tumor in the mediastinum. The gland may be enlarged during the course of leukæmia or of Hodgkin's disease.

Hemorrhages in the gland-tissue are not uncommon during scurvy and purpura hæmorrhagica.

IV. DISEASES OF THE DIGESTIVE SYSTEM.

1. DISEASES OF THE ŒSOPHAGUS.

ACUTE ŒSOPHAGITIS.

Etiology.—The œsophagus may be inflamed—(1) by the spread of inflammation from the pharynx or the stomach; (2) by mechanical or chemical irritants or corrosives; (3) by pseudo-membranous inflammation secondary to diphtheria or to some acute infections; (4) by the pustules of variola; (5) in rare cases œsophagitis may develop in sucklings without known cause.

Lesions.—The inflammation may be diffuse or localized, and either catarrhal, pseudo-membranous, or phlegmonous. The pustules of variola result in ulcerations. There may be a mycotic inflammation secondary to thrush and resembling it in its pathological features. The swallowing of corrosives is followed by sloughing and ulceration.

Symptoms.—Pain on swallowing is a nearly constant symptom, and a continuous substernal ache is frequently observed. Food may be regurgitated; if coated with blood or with pus, ulceration is indicated.

In cases of caustic poisoning the lips, the mouth, and the pharynx exhibit evidences of corrosion, and the symptoms of toxic gastritis are present, usually with some associated shock. Rupture of the œsophagus may occur. Patients recovering from the acute symptoms ultimately develop œsophageal stenosis.

Some cases of acute œsophagitis, even if severe, occasion but a trifling amount of discomfort.

The **treatment** of acute œsophagitis consists in the administration of the proper antidote in case of corrosive mineral poisons. Demulcent drinks and cracked ice are of service in diminishing the pain and the inflammation. The nourishment should be bland and unirritating. Fluids alone should be given during the acute stages, while in severe cases rectal alimentation should be insisted upon.

CHRONIC ŒSOPHAGITIS.

A chronic catarrhal inflammation of the mucous membrane lining the œsophagus is produced by improper and irritating food and by the presence of tumors or stricture of the œsophagus itself; or the chronic form may result from an acute attack.

The **symptoms** consist chiefly in the raising of mucus-coated regurgitated or vomited food.

The **treatment** is that of the underlying cause.

STENOSIS OF THE ŒSOPHAGUS.

Synonym.—Stricture of the œsophagus.

Stenosis may result—(1) *From compression from without* by tumor of the neck or the mediastinum, by aneurysm, by retropharyngeal abscess, or by a large pressure-diverticulum. (2) *From obstruction of the lumen* by foreign bodies, and rarely by tumors and polypi. (3) *From contraction of the wall.* (*a*) There may be *cicatricial contraction* following the healing of ulcers due either to corrosive poisons or to diphtheria, small-pox, or, more rarely, to syphilis or to tubercular disease. A rare form of ulceration, the "round ulcer," is seen at the lower end of the œsophagus. This ulcer is produced by self-digestion by regurgitated gastric juice, after the manner in which ulcer of the stomach is caused. (*b*) There may be *malignant growth* of the wall, usually epithelioma. (*c*) There may be *spasmodic contraction* or (*d*) *congenital narrowing* at some part.

Symptoms.—In all cases of stenosis of the œsophagus these cardinal symptoms are present—difficulty in swallowing, pain, and the regurgitation of food. The cases may, however, conveniently be described in three groups.

CICATRICIAL STENOSIS.

The stricture may occur at any part of the œsophagus, but it is most frequent in the lower third. The whole length may be involved. The stenosis may reach such a degree that liquids can barely trickle through. The œsophagus above the stricture is usually much dilated, and its walls are thickened.

After the history of antecedent ulceration the patient complains of increasing difficulty in swallowing, the food being cut finer and finer and washed down with water. In severe cases liquids alone are taken. The food seems to stick, and after a time it is regurgitated. The lower down the stricture is, and the more dilated the œsophagus above it, the longer the time after eating before regurgitation occurs. The ejected food may be macerated and mixed with mucus, but that it has not reached the stomach is proved by the absence of gastric odor and by the alkaline reaction of the food. The latter test, however, is not infallible if the food be retained some hours before being regurgitated, because of the formation of fatty acids in it. Pain is not a marked feature except at the time of swallowing the first bolus.

The **diagnosis** is made by the passage of the œsophageal bougie. A conical bougie on an elastic whalebone stem should be employed, but the soft-rubber stomach-tube may be used. It is of the utmost importance, before passing the bougie, to exclude aortic aneurysm producing stenosis by compression, because of the danger of causing rupture of the aneurysmal sac. The tube should never be passed when ulceration of the œsophagus from any cause is suspected. Auscultation is frequently serviceable in cases where the bougie cannot be employed. The auscultator, listening to the left of the dorsal spine while the patient swallows a mouthful of water, hears a loud splashing, gurgling sound at the site of the stricture, below which the sound is absent or only slightly audible after a pause.

The **prognosis** depends upon the degree of stenosis and upon its dilatability. In advanced grades death may ensue from inanition or from rupture of the dilated œsophagus above the stricture; or, should the regurgitated food enter the larynx, suffocation or aspiration-pneumonia may result.

Treatment.—Gradual dilatation by the persistent use of the œsophageal bougie should be employed, and in many cases the results are remarkably good. The diet should be compact and nourishing, and rectal alimentation may be resorted to. In advanced cases the stricture may be cut, or an opening may be made into the œsophagus below the stricture (œsophagostomy), or into the stomach (gastrostomy).

CANCEROUS STRICTURE.

This form of œsophageal stricture is usually primary. Epithelioma is most common; scirrhus and encephaloid are rare. The growth usually occurs in the lower third, next in frequency in the upper third, of the œsophagus. Beginning in the mucous membrane, it extends to form an annular constriction, usually involving one or two inches of the tube. Ulceration of the growth may occur, so that the stenosis becomes less marked, but the ulceration may extend and perforate the lung, the trachea, a bronchus, the mediastinum, the aorta, or the pericardium. Erosion of the vertebræ may occur. Secondary growths in adjacent lymphatic glands are common. The œsophagus above the cancer is usually dilated, and its walls are thickened. Epithelioma is seen in patients over forty years of age; it is more common in men than in women.

The **symptoms** resemble those of the cicatricial group in their essential features. Dysphagia is progressive, and it becomes so extreme that emaciation and inanition rapidly result. The regurgitated food frequently contains blood and pus in small quantities, and it may contain cancer-cells and fragments. Pain is a marked feature, being aggravated by attempts at swallowing. The cervical lymph-glands are enlarged, and symptoms of cancerous cachexia are present. The œsophageal bougie must be used with extreme caution to avoid penetrating the ulcerated wall, as such an accident has not infrequently occurred. The soft-rubber tube is generally preferable in these cases.

The **diagnosis** is made positive by the finding of cancer-fragments in the eye of the tube. In cases of ulceration of the epithelioma in which no real degree of stenosis longer persists, the tube may pass without difficulty. In these cases, however, dysphagia and regurgitation of food may be nearly as marked as if there were an actual narrowing of the lumen; these symptoms are to be explained on the theory that downward peristalsis is checked at the site of the growth, and that a reversed peristalsis results in regurgitation.

The **prognosis** is hopeless. Patients usually die, from inanition, perforation, or aspiration-pneumonia, about one year after the symptoms begin to be noticed.

The **treatment** consists in the proper feeding of the patient by nourishing liquid and concentrated food, by feeding through a stomach-tube or by the rectum. Gastrotomy or œsophagotomy offers but little chance even of prolonging life.

SPASMODIC STENOSIS (ŒSOPHAGISMUS).

This form of œsophageal stenosis occurs chiefly in young hysterical women or in those with marked neurotic tendencies. It may occur after an attack of choking, or as a nervous affection in those bitten by dogs and in dread of hydrophobia. It may occur from reflex causes, such as pregnancy, and it often complicates organic lesions of the œsophagus itself.

The **lesion** consists of spasm of the œsophageal wall, usually at either the pharyngeal or the cardiac extremity. In the former case it is often associated with spasm of the pharyngeal muscles.

The **symptoms** consist of inability to swallow, regurgitation of food, and a sense of substernal pain or constriction. The dysphagia comes on abruptly and is not progressive—two characteristics which distinguish the spasmodic from the other forms of stenosis. There are, moreover, periods of marked improvement; or the dysphagia may be only for certain articles of food, varying in individual cases. The inability to swallow is never so extreme as to endanger the life of the patient by inanition, although the disease may last for days, weeks, or even for months. Intermissions, however, usually mark the protracted cases. Associated hysterical or hypochondriacal symptoms are usually present, rendering the diagnosis the more evident.

The **prognosis** is perfectly good.

The **treatment** consists in passing the œsophageal bougie. Difficulty may be encountered by reason of the spasm, but patience and gentleness will usually succeed in accomplishing its passage. Often a brilliant cure follows the first treatment, but in obstinate cases a daily passage of the bougie may be needed, usually before the principal meal, to restore the confidence of the patient.

Sedatives, such as valerian, the bromides, and phenacetine, may be given, and tonic treatment is indicated in nearly every case.

DILATATIONS AND DIVERTICULA.

Dilatation may be primary or secondary.

Primary dilatation, which is rare, is due to a congenital defect in the muscular tissue of the œsophageal wall or to its paralysis. The œsophagus is enormously dilated and is usually longer than normal. The principal symptom is dysphagia from lack of sufficient peristalsis.

Secondary dilatation occurs with stenosis above the point of constriction. The condition is to be suspected if a patient with organic stricture of the œsophagus regurgitate large quantities of macerated food. The retention of such quantities of food may cause pressure-symptoms.

Diverticula are of two forms:

Pressure-diverticulum.—This form is most common at the posterior wall of the œsophagus, at its junction with the pharynx. From weakness of the muscle at this point a bulging of the mucous and submucous coats takes place, forming a hernial sac, into which food passes. Owing to lack of expulsive power, food collects and becomes macerated, the sac growing larger and larger. The sac may be emptied from time to time by contraction of the muscles of the neck or by external manipulation. It may be large enough, when full, to press forward and occlude the œsophagus.

The **diagnosis** is made by the presence in the neck of a tumor which can be emptied by manipulation, and by alternately passing the bougie down the œsophagus and into the sac.

Traction-diverticulum.—This form is situated on the anterior wall of the œsophagus, opposite the bifurcation of the trachea. Should the mediastinal glands normally present at this point become inflamed, they will enlarge and may become adherent to the wall of the œsophagus; by the subsequent contraction of the glands the wall is drawn out into a funnel shape, never more than a quarter of an inch in depth. This form gives no symptoms, although in rare cases perforation has been known to occur.

PARALYSIS OF THE ŒSOPHAGUS.

This rare condition develops from diseases of the brain and the cord, from hysteria, and occasionally as a post-diphtheritic paralysis.

The **symptoms** consist of difficulty in swallowing. The passage of the bougie reveals, however, no stricture. Paralytic dilatation may subsequently be developed.

The **treatment** consists in nourishing the patient by the stomach-tube, in treating the original cause, and in faradization of the œsophagus.

RUPTURE OF THE ŒSOPHAGUS.

This accident may occur during violent and sudden attempts at vomiting in healthy people, but it is exceedingly rare, rupture usually being due to the perforation of an œsophageal ulcer or of a foreign body.

The condition is fatal within a few days, and treatment is merely symptomatic.

VARIX OF THE ŒSOPHAGUS.

Varicose veins may develop in the lower portion of the œsophagus, as an evidence of congestion, of heart disease, or of cirrhosis of the liver. Chronic œsophagitis with vomiting of mucus usually results, and rupture of the varicose veins may lead to fatal hemorrhage.

2. DISEASES OF THE STOMACH.

ACUTE CATARRHAL GASTRITIS.

Etiology and Synonyms.—Among the causes predisposing to acute catarrhal gastritis may be enumerated lesions of the heart or of the liver causing chronic congestion of the stomach, and any condition of depreciated health or of fever that renders it difficult for the stomach to digest the food properly. Gouty individuals are apt to suffer from gastritis, and personal idiosyncrasy often plays an important rôle. The exciting cause is usually a dietetic error—overfeeding; eating when too tired to digest properly; food unsuitable for digestion, as hot bread, unripe fruit, or food improperly cooked. Over-indulgence in alcohol is a frequent cause. Severe attacks follow the taking of irritants or of tainted meat or fish, poisoned ice-cream, poor milk, or unripe fruit. Certain articles of diet, varying with each individual, may precipitate an attack. Gastritis is frequently symptomatic of an infectious disease or fever. *Synonyms:* Acute gastric catarrh; Acute indigestion.

Pathology.—The mucous membrane of the stomach is swollen, congested, and covered with tenacious mucus. There may be small submucous hemorrhages or small superficial erosions. The cells of the gastric tubules are swollen and cloudy, and the interglandular tissue may be infiltrated with leucocytes. Hydrochloric acid is usually temporarily absent from the gastric secretion, being replaced by lactic acid and the fatty acids. The most frequent seat of inflammation is near the pylorus, and the inflammation may extend to the duodenum or, especially in children, may involve the entire small intestine.

The **symptoms** are divided clinically into two sets of cases:

1. *Simple Gastritis* (*Acute Indigestion*).—The appetite is lost or is diminished except for highly-seasoned food. There are uncomfortable feelings referred to the stomach, in some cases amounting to severe colicky pain. Nausea is frequently complained of, and vomiting usually affords relief. The vomited matters, which consist of undigested food mixed with mucus and bile, are of an acid reaction from the presence of lactic and fatty acids. The patient complains of headache, depression, and prostration. The tongue is coated and the breath is offensive. The bowels are usually constipated, although diarrhœa may follow the attack. Fever usually is slight, although in some cases the temperature may reach 102° or 104° F. The abdomen is usually somewhat distended and tender in the epigastric region. Herpes, urticaria, or erythema may appear, especially in cases caused by eating shell-fish.

In young infants vomiting, fever, and prostration are the principal symptoms. In the symptomatic gastritis of infectious disease, vomiting and increased prostration are the prominent symptoms. The vomiting may be so excessive as to interfere with the nourishment of the patient.

2. *Gastritis from Ptomaïne-poisoning*.—This form of gastritis follows the eating of tainted meat or fish or of ice-cream containing the alkaloidal poison tyrotoxicon. Cases frequently occur in small epidemics among those who have eaten of some particular article of food. The symptoms are those of a severe gastritis, with marked prostration and incessant vomiting. In severe cases constitutional symptoms of an alarming nature appear; the pulse becomes rapid, the heart's action feeble, the skin becomes cold and clammy, and the patient is apt to die.

Treatment of Gastritis.—The first indication is to rid the stomach of whatever is irritating it. Nature often does this by the vomiting, otherwise a simple emetic is usually indicated. The bowels should be opened, castor oil or saline laxatives usually being employed for this purpose, although blue mass or calomel is often beneficial. A natural diarrhœa should not be checked. Should it be excessive and exhausting, a dose of castor oil (℥ss) and tincture of opium (♏ xv) in combination should be given. The diet should be light and easily digestible. These rules suffice for the majority of mild cases. In more severe cases the stomach should have a rest and food should be interdicted for a day, although cracked ice and carbonated waters may be given freely. When the vomiting is constant, rectal alimentation may be resorted to. Distressing symptoms should be controlled by appropriate medication. The vomiting should not be checked until the stomach is empty; after that bismuth in full doses, bicarbonate of soda, oxalate of cerium, or even small doses of codeia or of morphine, may be given. Pain is best relieved by emesis, by counter-irritation over the stomach, by poultices or mustard pastes, and by the administration of large doses of bismuth. Codeia or hypodermic injections of morphine are to be given only in severe cases. Stimulants may be indicated in the gastritis of ptomaïne-poisoning.

TOXIC GASTRITIS.

This form of gastritis follows the swallowing of concentrated acids, alkalies, or irritants, frequently taken with suicidal intent, or of certain non-corrosive poisons like phosphorus, arsenic, and antimony. In the former case the mucous membrane of the mouth, the œsophagus, and the stomach is marked with areas of necrosis surrounded by zones of intense inflammation, while the submucosa is hemorrhagic and infiltrated with serum. In severe cases perforation of the stomach may occur. In the non-corrosive poisons the process consists in fatty degeneration of the glandular elements, small-celled infiltration of the entire glandular connective tissue, and hemorrhage.

The **symptoms** are intense burning pain in the mouth, throat, and stomach, difficulty in swallowing, and constant vomiting, the vomited matters usually containing blood, and frequently containing portions of necrosed mucous membrane. The abdomen is distended and exquisitely tender. In very severe cases symptoms of collapse appear; the pulse is rapid and feeble, the skin is cold and clammy, and there is great prostration, frequently interrupted by restlessness or by convulsive movements. Albumin is usually present in the urine, and there may be hematuria. Perforation of the stomach is followed by death in collapse within a few hours. If the patient recovers there may result stricture of the œsophagus or extensive cicatrices in the stomach, leading to chronic atrophy and inanition.

The **treatment** is that of severe gastritis. Emetics should, however, not be given, on account of the danger of causing perforation. Siphonage of the stomach is preferable, a soft-rubber stomach-tube being passed with caution, and the stomach being washed with solutions of the appropriate chemical antidote. Hypodermic injections of morphine are needed to allay the pain and distress. Rectal alimentation is usually necessary; it should be resorted to in the severer cases.

ACUTE CROUPOUS GASTRITIS.

Synonyms.—Diphtheritic gastritis; Membranous gastritis.

Croupous gastritis may occur as a secondary infection with diphtheria, but is more common as a secondary process in pneumonia, typhus and typhoid fever, pyæmia, puerperal fever, and Asiatic cholera.

The **symptoms** are those of an intense gastritis together with those of the primary disease.

The **diagnosis** cannot be made during life.

The **treatment** is that of the severer forms of gastritis.

ACUTE SUPPURATIVE GASTRITIS.

Etiology and Synonyms.—This uncommon disease occurs more often in men than in women. It is rare as a primary disease, usually occurring after pyæmia, puerperal fever, or other septic diseases. *Synonyms:* Phlegmonous gastritis; Purulent gastritis.

Pathology.—The lesion, which consists in a suppurative process in the submucosa, presents itself in two forms—a diffused purulent infiltration, and a localized abscess; in the latter case rupture may occur into the stomach or into the peritoneal cavity.

The **symptoms** are those of gastritis and of a severe infection. There is severe pain in the stomach, usually with exquisite tenderness in the epigastrium. If the abscess be large, it may be felt externally. Vomiting is persistent and agonizing, and the vomited matters may contain pus. Jaundice has occurred in a few instances.

The symptoms of general infection are an irregular fever ranging between 102° and 105° F., rapid and feeble pulse, prostration, delirium, and finally coma. The disease may in rare instances run a subacute course, with pain, vomiting, irregular fever, and erratic chills.

The **diagnosis** is made with the utmost difficulty, especially in primary cases. Aid may be afforded if the abscess be large enough to be appreciated by the touch and if vomiting of a large amount of pus occur.

The **treatment** is simply palliative.

MYCOTIC AND PARASITIC GASTRITIS.

The invasion of the stomach by the bacillus of diphtheria and the pus organisms of phlegmonous gastritis has already been described. A fatal case of the growth of favus has been reported. The tubercle bacillus may involve the gastric mucous membrane, while sarcinæ and the yeast fungus are frequently found in cases of fermentation and in dilated stomachs, and serve to increase the inflammation.

Ascarides, tæniæ, earth-worms, maggots, and the larvæ of certain dipteræ have been found to be the cause of acute gastritis.

CHRONIC CATARRHAL GASTRITIS.

Synonyms.—Chronic dyspepsia; Chronic gastric catarrh; Atrophy of the stomach.

Etiology.—The causes of chronic gastritis are various and may be classified as follows:

Dietetic Causes.—Among these may be enumerated over-eating, over-indulgence in ice-water during meals, rapid eating, irregular meals, improperly cooked and unsuitable food, such as rich fried food, pastries, or hot bread, and the abuse of alcohol, tea, or tobacco.

Constitutional Causes.—Chronic gastritis may be produced by any debilitating disease which reduces nervous force or deteriorates the blood. The food, not being digested properly, is retained in the stomach, ferments, and sets up a chronic inflammation. In this way the gastritis is often associated with anæmia, chlorosis, tuberculosis, Bright's disease, diabetes, gout, and uterine disease.

Local Causes.—The disease may be secondary to lesions of the stomach, such as cancer, ulcer, or dilatation, or it may follow passive congestion of the mucous membrane as in cirrhosis, or in any obstruction to the portal circulation, chronic heart disease, and certain diseases of the lung producing general venous congestions.

Pathology.—Two forms of chronic gastritis are described, a simple and a sclerotic.

Simple Chronic Gastritis.—The mucous membrane is thickened, is grayish or congested in appearance, and is covered with thick tenacious mucus. The veins are usually congested, and patches of pigmentation from small submucous hemorrhages are common, especially near the pylorus. The membrane frequently has a granular or reticular appearance from irregular growth of connective tissue. The connective tissue and the muscular coats are usually also thickened, especially around the pylorus, and this thickening may be marked enough to cause a narrowing of that orifice. The gastric tubules are atrophied and deformed, their cells are cloudy and swollen, and between the tubules is an abundant small-celled infiltration. There may be cystic dilatation of individual tubules.

Sclerotic gastritis occurs as a final result of these parenchymatous and interstitial changes and presents two distinct forms:

In the first form the stomach is usually dilated and its walls are thinned. The mucous membrane is thin, smooth, and light grayish in color; its glandular elements undergo fatty degeneration and atrophy. In advanced cases nothing remains of the mucous membrane but a layer of round cells, a few cysts, and fibrous tissue. This process is also called "phthisis ventriculi" or "simple atrophy."

The second form, "cirrhotic atrophy," which is even more rare than the first form, shows the same glandular destruction, but the walls are much thickened by enormous overgrowth of muscular fibres and connective tissue, and the whole organ diminishes greatly in size. In rare cases the thickening may be so great as to be felt through the abdominal walls.

Symptoms.—Two clinical forms may be recognized, *simple gastritis* and *sclerotic gastritis*, of which the former is by far the more common.

Simple Gastritis.—Distress or oppression in the stomach is a fairly constant symptom; it may be slight, or less frequently it amounts to actual pain. It usually occurs after eating, but it may be more or less constant, and in some cases it is aggravated when the stomach is empty. In some cases the pain consists of a burning feeling under the sternum, due to over-acidity, usually from the presence of the fatty acids of fermenting food; this pain is generally associated with eructations of a sour, acid fluid. To this condition the names "pyrosis," "heartburn," and "cardialgia" have been given (although the term "pyrosis" has also been used as synonymous with "water-brash"). There may be tenderness over the stomach, more commonly diffused, and rarely severe. The appetite is usually impaired, although some patients complain of unnatural hunger. There may be appetite only for highly-seasoned and peculiar articles of food. Thirst is usually much increased. Flatulence is marked in the great majority of cases, producing distention of the abdomen, palpitation of the heart with intermittency of its action, a feeling of distress amounting at times to dyspnœa, and a stitching pain under the free border of the ribs. Relief is afforded by the eructations of gas, which may be odorless or offensive. There may be eructation of food or of fluid with the gas. The regurgitated food may be bitter or so acid as to set the teeth on edge; it may be associated with heartburn, or the fluid may be clear, watery, brackish, and of an alkaline reaction. This latter form, which is termed "water-brash" (or "pyrosis" of some authors), is most commonly observed in the gastritis of drunkards. It usually occurs in the morning, being due to an increased amount of saliva which has been swallowed during the night. Nausea and vomiting are not usually pronounced. Nausea, when present, usually occurs in the morning, and it is especially common in alcoholic cases associated with "dry retching." Vomiting usually occurs one or two hours after eating. The vomited matters consist of food in various stages of digestion and fermentation, mixed with mucus. Chemical examination shows diminution or absence of hydrochloric acid, and abnormal acids, such as butyric, lactic, and acetic acids, are present. Small quantities of blood, either bright red, or in brownish granules from alteration of its pigment, may be vomited from time to time. Digestion is usually much delayed, portions of food being present in the stomach as late as seven hours after eating, at which time a normal stomach should be empty. Absorption is slow, as is proved by the fact that iodide of potassium given in capsule is not found in the saliva in fifteen minutes, as is normally the case, but that it may not be in evidence for more than half an hour. The tongue is usually heavily coated and is indented by the teeth. The edges and the tip may be red, and in some cases the whole tongue has a "raw-beef" appearance. The breath is usually offensive, and a bad taste in the mouth is complained of, especially in the morning. Saliva and the pharyngeal secretions are usually increased, and the patient may complain of a cough, the "stomach-cough," probably due to pharyngeal irritation. The bowels are usually constipated, but attacks of diarrhœa with the passage of undigested food may occur. The urine may be diminished in quantity, of high specific gravity, and may deposit uric acid, urates, phosphates, or oxalate of lime. The patient loses flesh and strength in accordance with the gravity of the case; this symptom is the most reliable means of estimating the true extent of the inflammation. From the passage of undigested fermenting food into the small intestine, functional disturbance of the liver is induced, adding its symptoms. Among these symptoms may be cited hot and cold flashes, marked enough to suggest malaria to the patient, headache, dizziness, and inaptitude for mental and physical work.

Sclerotic Gastritis.—There is an entire absence of digestive power of the stomach. Hydrochloric acid, pepsin, and milk-curdling ferment are all absent, and the fasting stomach is usually empty. The symptoms are those of a severe dyspepsia with failure in nutrition. Vomiting is a prominent feature, and, associated with decided loss of flesh and of strength, might suggest the existence of cancer. In other cases a progressive anæmia is developed, the symptoms resembling those of pernicious anæmia. In cases of "cirrhotic atrophy" there may be inability of the stomach to hold more than a small amount of nourishment at any one time.

Prognosis.—The course is essentially chronic, with periods of marked improvement from time to time. The symptoms often come and go, depending on the general health and upon the thoroughness and efficacy of the treatment. A guarded prognosis for recovery should always be given, especially if the disease has lasted any length of time. Relapses may be induced by trifling indiscretions. The danger of atrophy must be considered in severe and long-continued cases, for this condition tends to shorten life by inanition and anæmia.

The **treatment** is (1) dietetic and hygienic, and (2) medicinal.

1. *Dietetic and Hygienic Treatment.*—Detailed attention should be paid to the correction of all dietetic errors that may seem causative of the disorder. As regards diet, the experience of each patient should be regarded, and care should be taken not to exclude too many articles of food, on account of the danger of undermining the general health. Over-eating, on the other hand, should be avoided. As a general rule, pastries, sweets, hot breads, and fried foods

should be excluded from the diet, and tea and stimulants are to be allowed only in moderation. Starches and sugars should be limited in cases with marked flatulence. The patient should not eat too hurriedly, nor should he eat heartily when too tired to digest properly, nor indulge in severe exercise after a full meal. Nibbling between meals and at night should not be permitted. Food difficult to digest, such as pork, hard-boiled eggs, young veal, and cabbage, should be avoided, and too much ice-water is not to be taken during the meals. Careful attention should be paid to the maintenance of good general health. The patient should have sufficient sleep, exercise, and fresh air. Anæmic and weakly conditions should be combated by appropriate medication. Strychnine is often of the greatest service in increasing the nervous and motor power of the stomach. Change of air and occupation and travel will frequently do more than all medicines combined.

2. *Medicinal Treatment.*—The indications are to increase digestive power and to check fermentation. To obtain the best results the patient should be watched and the effect of diet be observed. If there be difficulty in digesting the albuminoids, dilute hydrochloric acid and pepsin are indicated. The dilute acid is to be given in 15- to 20-drop doses after meals; and it is important in the use of pepsin to find a preparation that has actual digestive value, as most of the preparations in the market are worthless. Fairchild's essence of pepsin in 5-drop does is to be recommended. Pancreatin in doses of from 5 to 15 grains, with an equal amount of bicarbonate of soda, may be given one or two hours after meals; or tablets of pepsin and pancreatin in combination, although objectionable for theoretical reasons, may be given with benefit. Papoid in 1- or 2-grain doses may be given in tablets after meals; this drug has the advantage, as has pancreatin to a less extent, of digesting

both albumins and starches. If there be difficulty in digesting the starches, pancreatin or papoid may be used, or diastase may be given, especially if the stomach-contents be excessively acid. A teaspoonful of Forbes's diastase before meals acts efficiently in such cases.

Glandular activity of the stomach may be stimulated by the bitter tonics, quassia, gentian, columbo, cardamom, and condurango, preferably combined with alkalies and given well diluted before meals. This form of treatment is often of the greatest value.

Fermentation and flatulence may be controlled by attention to the diet and by the use of the preceding remedies. In obstinate cases antiseptics may be given after meals. Creosote, thymol, the essential oils, bismuth subgallate or salicylate, naphthaline, hydro-naphthol, and salicylate of sodium or salol may be used. The best results in the writer's hands have followed creosote (♏iij–iv) and subgallate of bismuth (gr. v–x) after meals.

If there be much mucus, especially in obstinate cases, systematic lavage should be resorted to. The stomach should be washed out every day or every second day, preferably the last thing at night or early in the morning, when the stomach is empty. A weak solution (2 per cent.) of bicarbonate of sodium or of boric acid may be employed in the more aggravated cases. Patients soon learn to wash out their own stomachs, and they are usually much pleased with the results. In cases not severe enough to demand lavage much good can be accomplished by drinking one or two glasses of hot water half an hour before breakfast, and possibly before each meal. Hot alkaline waters are even more effective. After drinking the hot water the patient should lie down for a quarter of an hour, if possible, on the right side. The bowels should be kept regular, and an occasional mercurial is often beneficial.

Distressing symptoms should receive appropriate treatment. Vomiting is rarely troublesome except in children. It may be controlled by bismuth, oxalate of cerium, creosote, drop doses of carbolic acid, nitrate of silver, dilute hydrocyanic acid in 2-drop doses, and chloroform. Acidity may be relieved temporarily by soda or magnesia.

DILATATION OF THE STOMACH; GASTRECTASIS.

Etiology.—The causes of dilatation of the stomach may be divided into three groups:

1. *Mechanical obstruction of the pylorus* from cancerous or cicatricial stenosis, from hypertrophy of the connective and muscular tissues, from congenital narrowing, from external pressure of a tumor, or from floating kidney. It should be remembered that stenosis may exist without dilatation, the stenosis being compensated by hypertrophy of the muscular coats.

2. *Abnormalities in the contents of the stomach.* Excessive eating and drinking may cause dilatation; such cases are common among insane and diabetic patients and among workmen in breweries who drink large quantities of beer. In rare cases acute dilatation has occurred from this cause. Fermentation of food generates gases which press upon and stretch the gastric wall, especially if it be weakened by inflammation.

3. *Impairment of muscular force.* This condition may occur with partial destruction of the gastric wall, as in cancer or ulcer; with chronic gastritis, especially the atrophic and sclerotic forms; with peritoneal adhesions; and with adynamic states of the general system, such as general debility, anæmia, tuberculosis, rickets, and illnesses of all kinds.

Pathology.—The various causes cited operate in one way—to cause a habitual increase of stomach-contents.

More food being put into the stomach than can conveniently pass the pylorus, the stomach becomes gradually dilated, so that it may reach an enormous size. In moderate cases the lower border of the viscus reaches the umbilicus, while in advanced cases it lies on the level of the anterior superior spines or even dips into the pelvis. If stenosis of the pylorus exists, the muscular tissue may be hypertrophied. The mucous membrane regularly shows the lesions of chronic catarrhal gastritis.

Symptoms.—Symptoms due to the primary cause often precede those due to the dilatation. The symptoms of chronic gastritis are present in every case, associated with the more characteristic symptoms of dilatation. Of these symptoms vomiting is the most characteristic. At intervals there is vomited a large quantity of liquid and food, as much as three or four quarts being vomited at one time. The vomita, which are sour from the presence of fatty acids, although HCl is diminished or absent, consist of undigested fermenting food and mucus often containing bacteria, yeast fungi, or sarcinæ ventriculi. On standing, the vomita separate into three layers: an upper, of a brownish froth; a middle, of a turbid fluid; and an under layer of food and débris. It is diagnostic for the patient habitually to vomit food taken the day previous. According to the size of the stomach and the degree of pyloric stenosis allowing more or less of the gastric contents to escape into the intestine, the stomach will be more or less rapidly over-filled with food and drink, and the consequent disengorgement leads to a certain periodicity in the vomiting. In very bad cases the stomach may toward the close of the disease lose its contractile power, so that vomiting ceases; this sign is to be regarded as of serious import.

Heartburn and acid eructations are frequent, from the fermentation of retained undigested food. There may be dragging feelings and a sense of weight from the mechanical effects of the dilatation. Pain and oppression are commonly observed. The bowels are usually constipated, although attacks of colicky pain and diarrhœa may occur, from the passage of the acid fermenting food into the intestine.

If stenosis of the pylorus is marked, little food or fluid may pass the obstruction. In consequence, loss of flesh and of strength become apparent, the patient is unnaturally hungry and thirsty, the urine becomes diminished in quantity and is concentrated, and painful cramps of the muscles may occur.

Physical Signs.—*Inspection.*—The abdomen is large and bulging, and the position of the greater curvature may be seen. In cases with pyloric stenosis waves of peristalsis or of antiperistalsis may be seen.

Palpation often reveals the peristalsis. If there be stenosis of the pylorus from simple or cancerous thickening, the mass may be detected. In many cases the boundaries of the stomach may be appreciated, the feeling of resistance being compared to that of an air-cushion. Bimanual palpation may elicit splashing sounds. These sounds, which are caused by the shaking together of air and water, may often be elicited in a healthy stomach after eating. To be of any diagnostic value, the splashing should be produced when the normal stomach should be empty—two hours after drinking, seven hours after a general meal. To facilitate palpation the stomach may be inflated. This should always be done with caution, and never to an extreme degree, especially if cancer or ulcer be supposedly present. The patient may be given gr. xx–ʒj of bicarbonate of sodium, followed by an equal amount of tartaric acid in water, and the inflated stomach can then usually be felt distinctly. A stomach-tube should be at hand for the withdrawal of the gas, should unpleasant symptoms occur. Another method is by passing a stomach-tube and pumping air through it by a Davidson syringe. The introduction of a stiff bougie so that its point can be felt through the abdominal wall is not a justifiable procedure.

Percussion.—The patient standing upright, two zones are to be distinguished—an upper zone of hyper-resonance, and a lower zone of dulness. If this dull zone be at or below the level of the umbilicus, dilatation may be suspected. To prove that the dulness is due to the presence of food or of fluid in the stomach, and not in the colon, the stomach may be siphoned of its contents, with the disappearance of the dulness.

It is important to remember that in women with lax abdominal walls the whole stomach may be displaced downward without being increased in size.

Auscultation.—Succussion is readily elicited, and a fine sizzling sound is frequently heard.

The **prognosis** in mild cases not due to pyloric stenosis is fairly good, a large number of cases being entirely cured, while in others the disease does not progress. If due to stenosis of the pylorus, the prognosis is bad, depending upon the extent and nature of the stenosis.

Treatment.—Attention to the diet is of the first importance, and without it neither a cure nor an improvement can be expected. The food should be simple, concentrated, and easily digested, and in bad cases should be taken in small quantities at frequent intervals. Carbohydrates and fatty food should be permitted only in the smallest amount, and liquids are to be partaken of sparingly. In advanced cases a resort to predigested foods and peptones for short periods is often of service. A cup of hot water before meals will allay excessive thirst more effectively than a larger amount taken with or after the meals. To relieve the dilated stomach of its accumulated contents the stomach should be emptied and then be washed clean with warm water or with weak alkaline solutions. If fermentation be active, a 1 per cent. solution of salicylic acid or of resorcin may be used. Lavage should be practised the last thing at night or early in the morning, and it should be repeated every day or every second day, according to the requirements of the case. By systematic lavage not only is the weight of the accumulated food removed, but the stomach is relieved of mucus and the irritating fatty acids of fermenting food, and the absorbent and secretory functions are stimulated. Strychnine is of great value, through its power to restore tone to debilitated muscular fibre; it is of special service in dilatation not depending upon pyloric obstruction. Iron and tonics are indicated to restore general systemic tone. Pepsin, papoids, pancreatin, and hydrochloric acid may be employed as digestive aids; antiseptics are useful if fermentation be active.

Faradization, either through the abdominal wall or by an electrode introduced through a stomach-tube, has been recommended. Massage from the fundus toward the pylorus is often beneficial. It should be employed daily, five hours after the principal meal. If there be bulging of the epigastrium, a cushion, a pad, or an elastic abdominal bandage should be worn.

In cases of stenosis of the pylorus operative procedures are often indicated. Gastro-enterostomy may be performed. In case the stenosis be cicatricial, Loreta's operation of stretching the pyloric ring, which has been followed by good results in the majority of cases, may be performed.

ULCER OF THE STOMACH.

Etiology and Synonym.—Ulcer of the stomach is found at post-mortems more frequently than it is diagnosed during the life of the patient, ulcer, either cicatrized or open, being found in 5 per cent. of persons dying from all causes, the scars being the more common. Females are more frequently affected than males, in the proportion of 3 : 2. The greatest liability is between the ages of twenty and forty years, but the disease is not uncommon up to the age of sixty. It rarely occurs in young children. It is more common in anæmic and chlorotic patients and those with menstrual disorders, and it is not infrequently associated with tuberculosis. It occurs especially among servant-girls and in shoemakers, in the latter case being consequent, possibly, upon the pressure on the stomach while at work. It is not as common in America as in Europe. *Synonym:* Peptic ulcer.

Pathology.—There is but one ulcer in 80 per cent. of all cases, but as many as 34 ulcers have been found. The usual situation (86 per cent.) is on the posterior wall near the pylorus, close to the lesser curvature. The ulcers vary in diameter from one-half to two inches, although they may be much larger. The usual shape is round or oval, although the larger ulcers are apt to be irregular, and several ulcers may coalesce to form one of an irregular shape. The ulcer has an oblique funnel shape, becoming smaller as it extends deeper, the successive ulcerated coats being distinctly terraced. The floor has a clean, "punched out" appearance, free from inflammatory changes, but in old cases the floor and walls of the ulcer may be indurated and thickened. The depth varies, the ulcer sometimes involving only the mucous coat, at other times extending to the deeper structures and even perforating the stomach-wall. The mucous membrane of the stomach shows almost regularly the lesions of a subacute or chronic catarrhal gastritis. Changes in the blood-vessels of the stomach have been found in a large proportion of cases. Among these changes may be mentioned embolism, thrombosis, and diffuse endarteritis of the arteries supplying the ulcerated area. Small aneurysms have been found in the floor of the ulcer. The mucous membrane of the stomach may show the lesions of subacute or chronic catarrhal gastritis.

Sequelæ and Complications of Ulcer.—These complications are of the utmost importance.

1. The ulcer may cicatrize. In some cases hardly any scar is left; in other cases, especially if extensive ulceration of the muscular coat has occurred, considerable puckering or deformity may occur. Cicatricial stenosis of the pylorus with dilatation of the stomach is a not infrequent result. It often happens that large ulcers may remain open for years without showing signs of healing.

2. The ulcer may perforate the wall of the stomach. Gastric contents enter the peritoneum, and a rapidly fatal peritoneal septicæmia follows. Perforation, which occurs in 6 per cent. of all cases, is more common with ulcers of the anterior than with those of the posterior wall.

3. Adhesions may form between the stomach and the surrounding viscera, especially the left lobe of the liver, the pancreas, and the omental tissues, nature "putting a patch on" to reinforce the weakened spot and to prevent perforation. Extension of the ulceration and secondary infection of pus microbes may lead to fistulous tracts and suppurating cavities in these adhering organs. Gastro-intestinal fistulæ are thus formed, and perforation into the pleura, the pericardium, and the left ventricle of the heart has been known to occur. The suppurative process may extend along the veins, causing a suppurative pylephlebitis with multiple abscesses in the liver. The adhesions may not be extensive enough to prevent perforation, but may suffice to shut the site of rupture from the general peritoneal cavity, so that a localized peritoneal abscess results. Perforation of the posterior wall produces an air-containing abscess in

the lesser peritoneal cavity, known as "subphrenic pyopneumothorax."

4. Erosion of a blood-vessel is of common occurrence. This accident may occur with recent acute ulcers, but it is more common in the chronic form with spreading ulceration. Ulcers on the posterior wall of the stomach may erode the splenic artery or the artery of the lesser curve.

Pathogenesis.—All authorities agree that gastric ulcer results from self-digestion of part of the stomach-wall by the gastric juice. Self-digestion is prevented in the normal stomach by the circulation of alkaline blood in the gastric mucous membrane. Aside from this, not much is known. The common occurrence of ulcer with anæmia and chlorosis, in which diseases a diminished alkalescence of the blood is present, would suggest that diminished alkalinity allowed of self-digestion; but this theory has serious objections. The generally accepted theory is that for some reason the circulation stagnates in a certain area of the stomach-wall, and, its nutrition failing, the part is acted upon and destroyed by the gastric juice. Experimental embolism of the gastric arteries has been followed by ulceration, and this experimental evidence accords with the funnel shape of the ulcer and with the actual post-mortem demonstration of an embolus or a thrombus plugging the nutrient artery in some cases. Local pressure or traumatism is supposed to act locally to produce changes in the circulation. Talma lately suggested that a cramp-like contraction of the stomach on full contents may cause such pressure on its wall as to interfere with the circulation and to allow of ulceration. By filling the stomach of an animal with gastric juice and ligating the orifice he found that, by causing firm contractions with faradism, ulcers resulted.

The formation of ulcers seems to be favored by hyperacidity and hypersecretion.

Symptoms.—There may be any or all of the symptoms of chronic gastritis. These dyspeptic symptoms may be trifling or of the most aggravated character, but to some extent they are almost invariably present. The distinctive symptoms are pain and tenderness, vomiting, and the vomiting of blood.

Pain is the most constant symptom. The characteristic pain is sharply localized in the epigastrium, frequently running through to the back. It is brought on by eating, usually within a few minutes, and it is aggravated by irritating food or by an excessive quantity of food. It ceases when the stomach is relieved of its contents either by the normal exit of food through the pylorus or by the act of vomiting. The pain is increased by exercise and is diminished by rest. There is usually a localized area of tenderness an inch or so below the ensiform cartilage. There may be, however, relief from pressure on the epigastrium. In many cases this classical pain is not present, variations being common.

(*a*) In some cases there is the ordinary pain of an associated gastritis.

(*b*) In other cases the pain does not occur until two or three hours after eating or until the stomach is empty ; the pain, being due to hyperacidity, is relieved by food.

(*c*) In some cases the pain comes on in attacks at intervals for weeks or months, and then the patient goes for a long time without attacks; the pains, however, return after a variable length of time.

(*d*) In other cases the pain is reflected to the nerves of the abdominal wall and has the characters of a neuralgia.

Vomiting.—The characteristic of the vomiting rests not with the act itself, but in the relief it gives to the pain. A pain after eating, vomiting, and relief is the regular sequence.

The vomited matters, which consist of undigested food, usually without evidences of fermentation, generally contain HCl in more than the normal quantity.

The vomiting with ulcer is not constant, and when it does occur it varies greatly in severity, some patients vomiting nearly everything they eat, others vomiting but seldom, if at all. In other cases the vomiting is due to the associated gastritis.

Vomiting of blood occurs in about one-half the cases. The vomiting may be slight, but more commonly it is profuse. If profuse and sudden, the blood raised is red, partly clotted, and unaltered. If less profuse, it may be retained in the stomach for a longer time, so that by alteration by the gastric juice it becomes black or brown, resembling "coffee-grounds." Repeated hemorrhages lead to a high grade of anæmia; sudden profuse hemorrhage may cause death. In most cases some blood, and in rare cases all the blood, passes the pylorus and is evacuated with the stools, giving them a black, tarry appearance. On adding water to these stools a more characteristic blood-color is developed.

In old ulcers with a thickened base an indurated mass may be felt on examination in the neighborhood of the pylorus. In these cases the symptons of dilatation of the stomach may be present.

Hysterical and anæmic symptoms are often present; frequently they may so overshadow the symptoms of the ulcer that the latter condition is overlooked.

Varieties in Clinical Course.—1. In some cases the disease runs a latent course and is found after death from some other disease, without having given symptoms during life.

2. Some cases run a latent course until there occurs a profuse hemorrhage which may terminate fatally or which may be followed by the symptoms of chronic ulcer.

3. With or without a brief history of gastric disturbance, sudden perforation occurs, causing speedy death.

4. In some cases the characteristic symptoms are not marked, but resemble those of chronic gastritis, so that a diagnosis cannot with certainty be made.

5. In some patients the disease shows periods of apparent cure, after which there is a return of the symptoms. It is hard to say whether fresh ulcers form or whether the cicatrix of an old ulcer becomes ulcerated.

6. In some cases the anæmia and cachexia from malnutrition become so marked as to suggest pernicious anæmia or cancer.

7. In other cases the symptoms of pyloric stenosis and secondary dilatation of the stomach are so marked that the symptoms of the primary ulcer are overlooked.

The **prognosis** is uncertain, as a fatal hemorrhage or perforation may occur at any time. The possibility of fistulæ and suppuration without the stomach, as noted under the heading of "Pathology," should be kept in mind. The duration of the disease varies from a few months to a number of years, the average being from three to five years. Relapses are frequent. Recent ulcers are more amenable to treatment than old, indurated ulcers. Death from hemorrhage occurs in 4 per cent. of all cases; from perforation, in 6.5 per cent. About 15 per cent. of all cases of ulcer of the stomach are fatal.

It is often hard to tell when the symptoms of ulcer end, as gastritis, neuralgia, or dilatation of the stomach due to the presence of the cicatrix may continue gastric symptoms.

Treatment consists in rest, attention to the diet, and the administration of drugs.

When the diagnosis is made, absolute rest in bed should be enforced until acute symptoms have subsided. The more absolute the rest the better the results. In chronic cases modifications of the rest-cure are permissible.

The diet should be regulated carefully and systematically. The food should be bland, easily digestible, and taken in small quantities at frequent intervals. The diet and rest should continue for at least three months. Milk (which may be peptonized), eggs, rice, sweetbreads, beef extracts, and milk gruel may be given at the start, with scraped beef, young chicken, and farinaceous puddings after a time. In very severe cases rectal alimentation should be resorted to for a few days, after which time it may be used to supplement feeding by the mouth.

Medicinal Treatment.—Lavage with weak alkaline solutions is serviceable in cases with hyperacidity, especially if vomiting be marked. It is also of benefit should gastritis or dilatation add its symptoms.

For the gastritis the ordinary methods of treating this condition may be tried.

For the pain, bismuth, oxalate of cerium, Hoffmann's anodyne, or dilute hydrocyanic acid may be used. Cold or hot applications to the epigastrium or counter-irritation by iodine, by blistering, or by a nitric-acid issue may be employed. Morphine is to be withheld because of the danger of forming the habit. If the pain be due to hyperacidity, alkalies with cannabis indica may be employed, and the stomach is to be washed out. If the pain be due to gastritis, lavage and antiseptics will frequently relieve it.

Vomiting is to be controlled by systematic lavage, by the use of bismuth, oxalate of cerium, and hydrocyanic acid, and by regulation of the diet. In severe cases rectal alimentation should be resorted to.

Hæmatemesis is to be treated with absolute rest and quiet of body and mind and by the giving of cracked ice. The patient should be put as rapidly as possible under the influence of opium, preferably by hypodermics. Drugs by the mouth, such as gallic acid, ergot, and acetate of lead, are of no service, and the efficacy of local applications of ice is doubtful. Stimulation should be moderate, as a depressed state of the circulation is nature's method of checking hemorrhage by allowing the formation of a thrombus.

After hemorrhage all food should be withheld from the stomach for several days.

Anæmic and cachectic conditions should be treated steadily and persistently by iron, arsenic, good fresh air, and a change of climate or of occupation if necessary.

CANCER OF THE STOMACH.

Etiology.—Next to the uterus, the stomach is the most frequent seat of primary cancer, the organ being involved in 21.4 per cent. of a total of over 30,000 cases. According to Welch, cancer of the stomach is the cause of death in 1 per cent. of all persons dying after the age of twenty years. It is almost always primary. The actual cause of cancer of the stomach is not understood. It occurs in men a little more frequently than in women, and it is more common in some races than in others, pure-blooded negroes being comparatively exempt. Three-fourths of all cases occur between the fortieth and seventieth years, but the occurrence of cancer between the ages of twenty-five and forty is not as exceptional as is often represented. The influence of heredity is seen in 14 per cent. of all cases. Chronic gastritis has preceded cancer in a fair number of cases, and carcinomatous changes in the wall of an old gastric ulcer have been described.

Pathology.—The varieties met with, in the order of frequency, are cylindrical-celled epithelioma, encephaloid, scirrhous, and colloid cancer. According to Welch, the pyloric region is involved in 60.8 per cent., the lesser curvature in 11.4 per cent., the cardiac end in 8 per cent., the posterior wall in 5.2 per cent., the whole or the quarter part of the stomach in 4.7 per cent., multiple tumors 3.5 per cent., greater curvature in 2.6 per cent., anterior wall in 2.3 per cent., fundus in 1.5 per cent.

Cancer begins in the mucous membrane of the stomach and grows in all directions. The portion of the growth projecting into the cavity of the stomach may be broad and flat or cauliflower-like, or ulceration may occur, allowing of hemorrhage or of perforation. Perforation into the peritoneum occurs in 4 per cent. of all cases, being usually prevented by the formation of adhesions between the stomach and adjacent structures. More rarely perforation occurs into the colon or through the abdominal wall, or fistulæ may be formed into the lungs, the pleura, and the small intestine. Ulceration is most common with encephaloid and cylindrical-celled epithelioma, less frequently in scirrhous and colloid cancer. Growths about the pylorus are apt to cause pyloric stenosis with secondary dilatation of the stomach. Growths in the cardia are apt to cause stenosis and secondary dilatation of the œsophagus. Chronic catarrhal gastritis almost invariably occurs with the growth of cancer.

Encephaloid cancer grows rapidly and forms cauliflower-like masses projecting into the cavity of the stomach, tending to ulcerate easily. It is soft, of a grayish-white or reddish-white color, and contains much blood. Microscopically the growth consists of a scanty stroma enclosing alveoli filled with irregular polyhedral and cylindrical cells. Metastases are common.

Cylindrical-celled epithelioma somewhat resembles the encephaloid, but is firmer, especially at the edges. It is prone to ulcerate and to form metastases. Cysts containing mucus are often found. This form of tumor consists of elongated tubular spaces filled with columnar epithelium with an abundant stroma, and it resembles the structure of tubular glands.

Scirrhous cancer may occur as a hard, circumscribed tumor or as a diffused thickening of the gastric wall. It is hard to the feel, and it is most often found at the pylorus, causing stenosis. Scirrhus has but little tendency to ulcerate. It consists of a hard fibrous stroma with relatively few and small alveoli.

Colloid cancer shows itself as an extensive uniform infiltration and thickening of all the layers of the wall of the stomach. It spreads with great frequency to involve adjacent structures, although actual metastasis is comparatively rare. It shows trabeculæ of connective tissue enclosing large alveoli filled with translucent colloid material. To this variety the name of "alveolar cancer" has been applied.

These varieties are often mixed in the same specimen, so that it is hard to say in what class the growth is to be placed.

Secondary cancerous growths occur by (1) *direct extension*, involving the œsophagus or any structure to which the stomach may become adherent, or by (2) *metastasis*. This latter is most frequent in the lymphatic glands in the neighborhood of the stomach; next in frequency in the liver, the peritoneum, the omentum, and the intestines; less frequently in the pleura and the lungs. The liver is involved in about one-third of all cases. The cervical and inguinal lymphatic glands are not infrequently involved, and occasionally growths occur subcutaneously near or at the navel.

The **symptoms** of cancer of the stomach may be described as gastric, constitutional, and those due to the secondary growths.

Gastric Symptoms.—(*a*) As chronic gastritis almost invariably accompanies cancer of the stomach, it gives rise to any or all of its regular symptoms. The appetite is usually more completely lost than in uncomplicated gastritis.

(*b*) *Pain* occurs in 92 per cent. of all cases, being rarely absent except in old people. The pain is severe, more or less continuous, and is described as burning, gnawing, or neuralgic. It may be referred to the epigastrium or between the scapulæ, but the locality of the pain is no indication of the seat of the growth. Pain is usually increased by eating, although this may not be evident until some little time after taking food.

The pain differs from the typical pain of ulcer (1) in being more continuous, so that the patient is often awakened at night by it; (2) in being less dependent on food; (3) in the increased pain being delayed some little time after taking food; (4) in being less sharply localized; and (5) in not usually being relieved by vomiting. There is generally tenderness on pressure over the growth, but the tenderness is less marked and less sharply localized than in ulcer.

(*c*) Vomiting occurs in from 80 to 88 per cent. of all cases. As a rule, it does not appear until the latter part of the disease, when the cancer has attained a considerable size, whereas the vomiting of ulcer comes on early. Vomiting is more frequent if either orifice be involved. If the pylorus be affected, vomiting occurs longer after eating than if the cardiac orifice be involved, the food often remaining for some hours in the stomach without being digested. There are cases, however, in which vomiting is an early and a distressing symptom, severe enough to cause a fatal termination. If the stomach be dilated, the vomiting may be typical of that condition. The vomited matters consist of undigested food, often fermenting and mixed with mucus. Fragments of ulcerating growths may be found, establishing a diagnosis. These fragments, however, are more frequently found in the washing out of the stomach. Free hydrochloric acid is absent in the great majority of cases of cancer of the stomach. As it is so frequently absent in other conditions, such as atrophy of the gastric mucous membrane, its absence is not diagnostic, but is merely corroborative if other symptoms of cancer be present. Its presence, however, is strong presumptive, although not absolute, evidence that cancer does not exist. To test for HCl, Ewald's test-breakfast, consisting of a Vienna roll and a cup of weak tea without milk or sugar, should be given on a fasting stomach. In one hour a soft stomach-tube should be passed and the gastic contents be expressed with gentle pressure over the stomach. After filtering, a drop of Günzberg's reagent should be added to each cubic centimeter of the filtrate. If HCl be present, a cherry-red color is developed and cherry-red crystals are precipitated. Günzberg's reagent, or the phloroglucin-vanillin test, consists of—

Phloroglucin,	2 grams;
Vanillin,	1 gram;
Absolute alcohol,	30 grams.

This liquid is yellow in color and should be kept in a dark bottle not exposed to the light. This test does not react to lactic acid.

(*d*) *Hemorrhages.*—It is important to distinguish between the slight and the copious hemorrhage of gastric cancer. An admixture of a small quantity of blood with the vomit is present in about half the cases. The blood is brown or black, resembling coffee-grounds, the normal pigment having been converted by the acids of the stomach into dark-brown hæmatin.

Copious hemorrhages occur in about one-eighth of the cases. The blood may be bright red or more or less darkened, according to the length of time it has been retained in the stomach. Following profuse hæmatemesis some black tarry blood is usually passed with the stools.

Hemorrhage in cancer differs from that in ulcer (1) in being more frequent; (2) in being usually less copious; (3) in being retained longer in the stomach, with the consequent "coffee-ground" appearance; and (4) in occurring in the later stages, when the cancer is ulcerating and the patient is emaciated and cachectic, whereas in ulcer the hemorrhage is an early symptom, occurring when the patient is apparently in good health.

(*e*) If dilatation of the stomach or stenosis of the cardiac end of the œsophagus complicate, the symptoms of these conditions will be added.

Constitutional Symptoms.—The patient grows progressively anæmic, developing a waxy or "beeswax" pallor which is quite characteristic; emaciation and prostration become more and more marked; there may be a slight irregular rise in the afternoon temperature. Slight œdema of the ankles is common, and a general itching of the skin is complained of—a symptom which in old people should suggest cancer or diabetes. In rare cases chills with paroxysmal elevations in temperature have been recorded. Delirium or coma may occur as a terminal event.

Symptoms of the secondary growths vary according to their size and location. Metastases in the liver cause a general enlargement of that organ, with pain, tenderness, and jaundice. Ascites is not uncommon. Involvement of the peritoneum and the omentum gives rise to ascites and abdominal pain and tenderness, and on examination the thickened masses may be evident. The glands above the clavicle are often enlarged. The subcutaneous nodules near the umbilicus have been alluded to.

Physical Examination.—Owing to the position of the stomach, only tumors of the pylorus, of the anterior wall, and of a large part of the greater curvature are accessible to examination. Tumors of the fundus, of the posterior wall, and of the greater part of the lesser curvature cannot be detected unless of some considerable size or unless the stomach be displaced downward. Tumors of the cardiac end cannot be detected at all.

As the growth is usually at or near the pylorus, the tumor is in most cases felt in the epigastric region as a firm, hard, nodular mass, tender on pressure. It is not influenced by respiration unless adhesions with the liver are present. Owing to the weight of the growth dragging the pylorus downward, the tumor may be felt as low down as the iliac region. A definite tumor is felt in 80 per cent. of all cases. Examination may be made with the patient lying down, with the knees drawn up so as to relax the abdominal wall; in doubtful cases examination in the knee-chest position may be more satisfactory. Inflation of the stomach with gas (see Dilatation of the Stomach) may be resorted to; this procedure frequently brings the tumor into reach. In cases of colloid cancer no regular tumor-mass may be felt, but the stomach-wall feels abnormally thick and resistant. It is rare to feel a tumor before the third to the sixth month of the disease.

Peculiarities in the Clinical Course.—1. Some cases run a latent course, death resulting from some intercurrent disease. This occurs especially in aged subjects.

2. In some cases gastric symptoms may be insignificant, while anæmic and cachectic symptoms are marked. These cases are often diagnosed as pernicious anæmia or Bright's disease.

3. In other cases gastric symptoms are developed, but are not characteristic. These cases are often diagnosed as chronic gastritis or dilatation.

4. There are cases in which the symptoms of the primary gastric cancer are obscured by symptoms arising from the secondary growths. These cases are then diagnosed as primary cancer of the liver or of the peritoneum.

The prognosis is practically hopeless. The duration of the disease is usually about two years. Cases live about one year after the diagnosis is made. Occasionally may be seen cases with a rapid course terminating in from three to six months.

Treatment.—If the growth be localized and non-adherent to other structures, and if no secondary deposits are present, surgical interference may be justifiable. Resection or gastro-enterostomy may be performed, although the results as yet are far from encouraging. Medicinal treatment, on the other hand, is merely palliative. Condurango at one time was lauded as a specific, but it is now recognized only as an excellent stomachic tonic, in some cases relieving the pain and the vomiting. It may be given as a decoction, a wine, or a fluid extract. Hydrochloric acid is indicated as an aid to digestion, and it is usually of service. The pain and the vomiting are to be controlled on general principles of treatment. As the disease is certainly fatal, there can be no objection to giving opium, as it makes no difference whether or not the habit is formed.

NON-CANCEROUS TUMORS OF THE STOMACH.

These growths are rare and are not causative of destructive symptoms.

Polypi are the most common form. They are composed of hypertrophied mucous membrane, are rarely larger than a bean, and are usually multiple.

Sarcomata are rare. *Fibromata* and *lipomata* are occasionally met with. *Lymphomata* may be found in connection with leukæmia or pseudo-leukæmia. *Myomata* or *fibromyomata* may occur in the form of large polypi. Cases of foreign bodies have often been mistaken for tumors. The most remarkable instances are the hair balls in hysterical women who are in the habit of eating their own hair.

HEMORRHAGE FROM THE STOMACH.

Synonyms.—Hæmatemesis; Gastrorrhagia.

Etiology.—The causes of hemorrhage from the stomach are exceedingly various; they may be grouped as follows:

1. *Traumatism.*—(*a*) Mechanical injuries, as blows or falls; penetrating wounds or the rough introduction of a stiff stomach-tube. (*b*) Chemical injuries by strong acids or alkalies or by corrosives.

2. *Local Disease of the Stomach.*—(*a*) Ulceration from ulcer or cancer. (*b*) Disease of the blood-vessels, such as miliary aneurysm, atheromatous or fatty degeneration, or varices of the veins.

3. *Congestion of the Gastric Mucous Membrane.*—(*a*) Active congestion. This condition may occur with acute gastritis. Under this heading may be included vicarious menstruation by way of the stomach. (*b*) Passive congestion. This condition occurs with cirrhosis of the liver, thrombosis of the portal vein, or pressure on the vein by a tumor. It occurs secondarily with chronic diseases of the heart or of the lungs attended by general venous congestions. Congestion of the stomach often arises from splenic enlargement, being explained by the intimate relations between the vasa brevia and the splenic circulation. Congestion occasionally occurs during the expulsive efforts of parturition.

4. *Disorganized Blood-conditions.*—(*a*) Hæmatemesis may occur with any of the severe infectious diseases, especially yellow-fever, small-pox, measles, scarlet fever, relapsing fever, malaria, and typhus fever. In these cases it is usually associated with other hemorrhages from mucous membranes and under the skin. (*b*) Toxic conditions, such as cholæmia and poisoning from phosphorus. (*c*) Hemorrhagic diseases. Among these diseases are to be mentioned scorbutus and

purpura hæmorrhagica. Under this heading may be inserted hæmophilia. (*d*) Profound anæmia, whether idiopathic, malarial, leukæmic or pseudo-leukæmic, or due to Addison's disease.

5. *Nervous Causes.*—There are cases of hæmatemesis not infrequently occurring in hysterical subjects without assignable cause. The disease is not uncommon with progressive paralysis of the insane and with epilepsy.

6. *Idiopathic Hemorrhage.*—This condition was described by Flint as hemorrhage occurring, as it often does from the nose, without assignable cause, and not being due to any morbid condition.

7. *Melæna Neonatorum.*—This is a severe and usually fatal hemorrhage occurring in infants within the first two weeks of life. It may occur in healthy children or in those with a family history of hæmophilia, or it may occur in prematurely born children from too early an interruption of the fetal circulation. In some cases it depends on gastric ulcer.

8. The blood may not come primarily from the stomach, but may flow into it. In hæmoptysis, epistaxis, or bleeding from the throat blood may be swallowed, to be vomited later. Blood may trickle down from the œsophagus from rupture of varicose veins in its wall, from ulceration, or from rupture into it of a neighboring aneurysm. The blood may enter the stomach from rupture into it of an aneurysm of the abdominal aorta or one of its branches, or ulceration may perforate the heart or the lungs, allowing of hæmorrhage in this way. Nursing babies may swallow blood from the mother's breast, as from cracked and bleeding nipples. Hysterical patients often swallow blood from slaughter-houses, and vomit it to obtain the sympathy of friends.

The **symptoms** consist of hemorrhage and anæmia. In rare cases the patient may die before any blood has been vomited or passed with the stools. In all cases of profuse hemorrhage some blood, and in rarer cases all the blood, may be passed by the bowels, giving the stools a black, tarry appearance. Water added to such stools will develop a more characteristic blood color. The amount of blood lost may amount to three or four pounds in the course of a single day. Anæmic symptoms are marked in proportion to the amount of blood lost.

Diagnosis.—Careful inspection usually leaves no doubt that it is blood that is vomited. Should doubt exist, recourse may be had to the microscope, the spectroscope, and the test for hæmin-crystals. The diagnosis from hæmoptysis is to be based upon the points contained in the following table, compiled by Welch:

Hæmoptysis.	*Hæmatemesis.*
1. Usually preceded by symptoms of pulmonary or cardiac disease. Bronchial hemorrhage without evidence of preceding disease is not rare.	1. Usually preceded by symptoms of gastric or hepatic disease, less frequently by symptoms of other diseases.
2. The attack begins with a tickling sensation in the throat or behind the sternum. The blood is raised by coughing. Vomiting, if it occurs at all, follows the act of coughing.	2. The attack begins with a feeling of fulness in the stomach, followed by nausea. The blood is expelled by vomiting, to which cough, if it occurs, is secondary.
3. The blood is bright red, fluid or but slightly coagulated, alkaline, frothy, and frequently mixed with muco-pus. If the blood has remained some time in the bronchi or in a cavity, it becomes dark and coagulated.	3. The blood is dark, often black and grumous, sometimes acid, and usually mingled with the food and other contents of the stomach. If the blood is vomited at once after its effusion, it is bright red and alkaline; or it may be alkaline if it is effused into an empty stomach.
4. The attack is usually accompanied and followed by localized moist râles in the chest, and there may be other physical signs of pulmonary or cardiac disease. Bloody sputum continues for some time, often for days, after the profuse hemorrhage ceases.	4. After the attack the physical examination of the lungs is usually negative, but there are generally symptoms and signs of gastric or hepatic disease. Black stools follow profuse hæmatemesis.

The treatment of hæmatemesis is considered under Gastric Ulcer, page 462.

NEUROSES OF THE STOMACH.

Nervous Dyspepsia.

Under this heading are included those cases of dyspepsia in which the stomach is apparently healthy and in which digestion is performed within the physiological time-limit.

Etiology.—The condition is met with in those who are overworked, nervous, and worried, and in hysterical and neurasthenic patients.

Symptoms.—*Gastric symptoms* resemble those of chronic gastritis in the main, but are less localized; vomiting rarely occurs, the symptoms are less dependent upon eating, and the tongue is less apt to be coated than in chronic gastritis. Chemical examination of the gastric juice allows of classification into three chief types: Nervous dyspepsia (1) with normal secretion, (2) with diminution of hydrochloric acid, and (3) with the condition of hyperacidity or hypersecretion. The characteristic gastric symptoms of the last type are discussed under a separate heading.

Intestinal symptoms consist of flatulence and irregularity in the action of the bowels.

Nervous symptoms antedate the dyspepsia and are aggravated by it, a vicious circle being thus established. The general condition is one of neurasthenia of any grade of severity. Hypochondriasis is almost always present.

The **diagnosis** of the nervous form of dyspepsia is to be made by the exclusion, so far as possible, of organic disease of the stomach and by the presence of a marked neurasthenic element. It is often difficult to differentiate between chronic gastritis with resulting neurasthenia and a primary neurasthenia with the symptoms of nervous dyspepsia. In the majority of cases the diagnosis is rather a matter of professional instinct.

The **prognosis** depends upon the curability of the underlying neurasthenia.

Treatment is to be directed to the general condition. Patients are apt to over-diet and to run themselves down. This should not be permitted, but a liberal, wholesome diet should be insisted upon. In obstinate cases with well-marked neurasthenic symptoms the Weir Mitchell rest-cure often yields brilliant and permanent results.

HYPERSECRETION AND HYPERACIDITY.

Under these terms are included cases of gastric distress due to an excessive secretion or an increased acidity of the gastric secretions. Normally the gastric juice is secreted only when required for digestion, but in hypersecretion the process continues during fasting. Hypersecretion may be continuous or periodic, the latter form being usually associated with locomotor ataxia or with profound neurasthenia. Hyperacidity is more commonly observed, and is one of the complications of gastric ulcer.

Symptoms.—Pain, which is of a gnawing, burning character, is referred to the epigastrium or the heart, or it may be substernal. It is often spoken of as "heartburn." There are eructations of an acid fluid setting the teeth "on edge," and vomiting of a clear acid fluid may occur. Pain and acid eructations may occur with acid fermentation of the contents of the stomach, but the symptoms occur soon after eating, are not relieved by eating, and the eructated fluid contains fermenting particles of food; whereas the symptoms of hypersecretion or of hyperacidity occur when the stomach is empty, are relieved by eating, and the eructations consist of fluid alone. Digestion is usually retarded, especially for the starches.

The **diagnosis** of hypersecretion may be made by finding an acid secretion in the stomach during fasting hours. Hyperacidity is determined by the quantitative analysis of the gastric juice, obtained through a stomach-tube one hour after an Ewald test-breakfast consisting of a Vienna roll and a cup of weak tea without milk or sugar.

Treatment.—Neurasthenic conditions demand appropriate treatment. Alkalies in large doses (sodium bicarbonate, ʒj–ij) should be given two or three hours after meals, and may be combined with cannabis indica (gr. $\frac{1}{3}$, Herring's English extract). This combination not only relieves the pain and distress, but seems to have a decided curative effect. In obstinate cases a strictly meat diet may be enforced, or the stomach may be washed out with a weak alkaline solution every day before the principal meal.

GASTRALGIA ; GASTRODYNIA.

Neuralgic pain in the stomach may occur as a symptom of organic disease, as ulcer or cancer, or in the form of the "gastric crises" of locomotor ataxia. Aside from these cases, gastralgia may occur as a functional neurosis, independent of organic disease, and to this form the term "gastralgia" should properly be confined.

Etiology.—Most patients are neurasthenic, hysterical women, usually those at the change of life, but young women and men may also be affected. The attack seems to be favored by a gouty tendency, malarial poisoning, hysteria, and hyperacidity or hypersecretion. Gastralgia is one of the regular symptoms of chronic poisoning by lead. The attacks may be induced by excessive chewing of tobacco or by over-indulgence in tea.

Symptoms.—The disease appears in attacks of gastric pain, of a burning, tearing, or boring character, usually referred to the epigastrium, and occasionally radiating to the back and around the waist. Light pressure is grateful, but tenderness is usually elicited by firm pressure. Vomiting is rare. Relief is frequently afforded by eating. During the attack the abdomen is usually retracted, and the patient assumes a generally flexed position. The attacks, which last from a few minutes to several hours, may be repeated at irregular intervals, or they may occur with such periodicity as to suggest a malarial influence. They may recur at night, and they are usually independent of eating, although in some cases pain may arise from dietetic causes. The symptoms of gastralgia are usually associated with those of a neurasthenic or hysterical nature, giving a decidedly neurotic stamp to the majority of cases.

The **diagnosis** is to be made by the exclusion of organic lesions and by the general view of the case, showing admixture of neuralgic and neurasthenic symptoms. The diagnosis is often made with great difficulty.

The **prognosis** depends upon the curability of the underlying cause.

Treatment.—For the attack itself, hot applications or a mustard paste may be applied to the epigastrium. Carminatives are often of service—Hoffmann's anodyne, spirits of chloroform, or valerian. Morphine is not to be used, on account of the possibility of a habit being formed; while cocaine is not recommended, because of its extreme depressant effect. Relief is frequently afforded during an attack by copious draughts of hot water. To prevent recurrences the underlying cause should be detected and removed. Neurasthenic conditions require appropriate treatment. Arsenic in free doses, but not to the point of tolerance, frequently acts as a specific. The combination of valerianate of zinc (gr. iij) with small doses (gr. $\frac{1}{8}$) of nitrate of silver is often useful. Hyperacidity and hypersecretion, if present, require their special treatment.

NERVOUS VOMITING.

Nervous vomiting does not arise from organic disease, but is a pure motor neurosis depending upon cerebral or reflex irritation.

Etiology.—Nervous vomiting may accompany lesions of the brain, the cord, or the meninges, hysteria, neurasthenia, or migraine. It may be due to reflex irritation from lesions of the abdominal or pelvic organs, and it occurs with sea-sickness and pregnancy. In neurasthenic males nervous vomiting may be due to irritation of the genito-urinary organs. The gastric crises of locomotor ataxia are described elsewhere. The "periodical vomiting" of Leyden is associated with gastric pain; it occurs in anæmic and nervous patients.

The **symptoms** of nervous vomiting differ from those of ordinary vomiting in that nausea and retching are seldom observed. It is rather a regurgitation than a vomiting. Food is usually ejected after meals, but the vomiting, which may occur at irregular intervals, is so little dependent upon dietetic errors that the name "causeless vomiting" is often applied to these cases. In hysterical cases, although all food may apparently be vomited, the general nutrition may remain good.

Treatment is to be directed to the underlying cause. Change of climate is often of great service, especially in cases of the primary periodic vomiting of Leyden.

PERISTALTIC UNREST.

Peristalsis of the stomach is increased, with the production of loud splashing sounds often heard at a considerable distance. This condition is not uncommon in neurasthenic subjects; it is usually increased by emotions.

RUMINATION; MERYCISMUS.

Hysterical and feeble-minded patients may regurgitate the food and chew the cud like ruminating animals. The habit is frequently hard to cure, but it seems to exert no evil effect upon the general health.

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3. DISEASES OF THE INTESTINES.

MORNING DIARRHŒA.

The etiology and pathology of morning diarrhœa are unknown. It is probable that the disease is functional and not inflammatory.

The **symptoms** consist of diarrhœa, usually limited to the early morning hours. There may be but one passage, or the diarrhœa may continue throughout the forenoon. The passages are usually painless and are accompanied by the passage of flatus. The diarrhœa may be varied by periods during which the bowels are normal or constipated. Mental worry is usually associated with the disease, and exhaustion may occur should the diarrhœa be excessive. In protracted cases the diarrhœa begins at an earlier hour of the morning, so that patients may be awakened at four or five o'clock in the morning with pain and an urgent desire for stool. In long-continued cases there may be developed a sense of impending movement of the bowels whenever any food is taken into the stomach.

Treatment.—A change of climate is frequently followed by a most brilliant result, which in the majority of cases is permanent. The general health should be built up; various modifications of diet should be tried, such as diets from which the starches and sugars are excluded, or a diet of meat alone. The treatment by drugs is not satisfactory, but salol, naphthaline, and the subgallate of bismuth may be used. Delafield finds his best results to have followed castor oil in doses of from 5 to 10 drops.

ACUTE CATARRHAL ENTERITIS.

Synonyms.—Acute ileo-colitis; Acute entero-colitis; Acute intestinal catarrh; Acute diarrhœa.

While certain portions of the small intestine may be inflamed more than others, it is not usually possible during life to say which portion is especially involved. In the great majority of cases the small intestine throughout its length is affected, together with the upper portion of the large intestine, and to this condition the names of "enteritis" and "entero-colitis" are applied.

Duodenitis causing catarrhal jaundice will be considered under the latter heading.

Etiology.—The causes may be primary or secondary.

Primary Causes.—1. Error in food, either in quantity or in quality. The commonest cause is the ingestion of unripe fruit or of food or milk in which decomposition-changes have taken place before its ingestion. Individual peculiarities play a considerable part in the causation of the disease, for what is food for one may be poison for another.

2. Impurities in drinking-water often cause epidemics of enteritis. Strangers are more susceptible to such impurities than are those who are accustomed to the water.

3. Toxic causes, such as irritant food or drugs, either alkaline, acid, or corrosive.

4. A sudden fall in temperature or the chilling of the surface after excessive perspiration may induce an attack.

5. Changes in the intestinal secretions may theoretically give rise to conditions leading to catarrh, but of these changes we know practically but little.

Secondary Causes.—1. Enteritis is often secondary to any gastric cause allowing fermenting or undigested food to pass into the intestine.

2. Peritonitis or any organic disease of the intestine, such as ulcer, hernia, or cancer.

3. Enteritis is favored by any chronic congestion of the intestinal blood-vessels from chronic heart or lung disease, or by any cause producing obstruction in the portal circulation.

4. Enteritis often occurs in the course of acute infectious disease; it may attend chronic cachectic conditions, such as those occurring with cancer, tuberculosis, Bright's disease, or anæmia.

Pathology.—The mucous membrane is red, swollen, congested, and covered with mucus. These pathological appearances often disappear after death, leaving the mucosa pale and sodden. The solitary and agminated glands are swollen and prominent, especially in children. Follicular and catarrhal ulceration may occur.

Symptoms.—There is pain of a colicky character, which may be diffused or may be localized at the umbilicus. It is usually worse before an evacuation, and is generally relieved by firm pressure. A tendency to straining indicates that the lower portion of the colon is involved. Tympanites and gurgling noises or borborygmi usually accompany the attack, being due to the presence of fluid and gas within the intestine and to increased peristalsis. If the inflammation be confined to the small intestine, there need be no diarrhœa. If the colon be involved, there is a loose fecal diarrhœa, the thin, gruel-like stools often containing portions of undigested food (lienteric diarrhœa) and flakes of brownish mucus. The color of the stools varies from dark brown to yellow, or even to gray, according to the amount of bile with which they are mixed. The number of evacuations varies from three or four up to twenty in the course of the day. There is usually loss of appetite, with occasionally nausea or vomiting. Fever may be absent, or there may be a rise of temperature of a few degrees.

The **prognosis** is perfectly good, the attack lasting from five days to a week and terminating in recovery. Relapses are frequent from repetitions of the original exciting cause.

Treatment.—In mild cases rest and a restricted diet suffice. The dietetic rules to be observed in chronic gastritis are applicable to these cases. In more severe cases a milk diet during the acute attack may be indicated. The patient should be kept warm, and a flannel band over the abdomen is of service, particularly in children. A cathartic is usually indicated at the onset, despite the existence of diarrhœa. The best drug for this purpose is castor oil or calomel. It is not wise to check diarrhœa for forty-eight hours. After this time astringents may be given, combined with opium in small doses. Bismuth subnitrate in gr. xx–xxx doses every two hours is usually efficient. Pain may be controlled by hot applications to the abdomen, by small doses of opium, or by spirits of chloroform in ʒss doses.

CHRONIC CATARRHAL ENTERITIS.

Etiology and Synonyms.—The most frequent cause of chronic catarrhal enteritis is the continuance of improper food and hygiene. It may follow repeated acute attacks; it follows chronic congestion of the portal circulation, chronic lung and heart diseases, and diseases of the liver, such as cirrhosis or pressure on the portal vein. It may occur with chronic lesion of the intestine, such as cancer or tubercular ulceration. Cachectic and debilitated conditions predispose to the disease, and it seems to be more common in those with a gouty tendency. *Synonyms:* Chronic intestinal catarrh; Chronic diarrhœa; Chronic catarrhal enterocolitis.

Pathology.—The lesion is a chronic catarrhal inflammation of the small, and usually of the large, intestine. The mucous membrane is generally congested and covered with mucus, and the wall of the intestine is thickened by hypertrophy of all its layers. In other cases the wall of the intestine is thinned, the glandular elements undergo atrophy, and the mucous membrane is grayish or lead-colored. The lymph-follicles are swollen and pigmented. Pigmentation of the villi also occurs. Catarrhal or follicular ulceration is seen in severe cases, the latter form being especially well marked in the lymph-follicles of the descending colon and the sigmoid flexure. The close approximation of these conical ulcers often gives to the colon a sieve-like or honeycombed appearance. These ulcers may perforate or be the seat of hemorrhage.

The **symptoms** of chronic catarrhal enteritis resemble those of acute entero-colitis, but are more protracted. As the large intestine is almost regularly involved, diarrhœa is a prominent symptom in most cases. In other patients diarrhœa alternates with periods of constipation. The stools usually are thin and fecal, containing undigested food mixed with mucus. If mucus be evenly admixed with the stool, its source is probably the small intestine; if it coat the stool, it comes from the large intestine. The patient frequently passes lumps or strings of glairy mucus, which may comprise the entire stool. Blood or pus may be present in the dejecta, the presence of the latter being a sure indication of the existence of ulceration. The number of stools varies from one to eight in the course of the day. In some cases the diarrhœa occurs in the early morning hours; in other cases it is induced by eating.

Pain to some degree is usually present. It may be diffused, or localized at the umbilicus and of a colicky character. It is most common in from one to three hours after eating. In other cases there is only a sense of oppression and fulness. Borborygmi and flatulence accompany the disease, and there may be symptoms of an associated gastritis or of functional disturbance of the liver. In aggravated cases the general health fails, the patient becoming thin and weak, and the emaciation and prostration may be extreme. Hypochondriasis or melancholia may occur.

The **prognosis** for perfect recovery is usually bad, although much may be done to relieve the patient. There are usually periods of temporary improvement, even if the case be not treated. The disease may be fatal in debilitated and aged patients and in children. The rare accident of perforation should not be forgotten.

Treatment should be carried out faithfully and patiently. The diet should be regulated to suit the peculiar needs of each case. As a rule, starches, sugars, and fats are objectionable. In aggravated cases recourse may be had to a meat or an exclusive milk diet. The bowels should be regulated. If constipation exists, cascara, podophyllin, aloin, or compound extract of colocynth may be used. A good combination to be recommended is the following:

℞. Strychninæ sulphatis,	gr. $\frac{1}{40}$;
Resin. podophylli,	gr. $\frac{1}{12}$;
Ext. belladonnæ,	gr. $\frac{1}{4}$;
Pulv. ipecac.,	gr. $\frac{1}{4}$.—M.

Ft. pil. No. j.
Sig. Such a pill after each meal.

Massage or faradization of the abdomen may be employed. If diarrhœa is present, astringents with intestinal disinfectants are indicated. Among these may be mentioned subnitrate of bismuth, ʒss-j; subgallate of bismuth, gr. x-xx; naphthaline, gr. x–xv; salicylate of bismuth, gr. x–xx; or salol, gr. v–x,—these doses being repeated three or four times in the day. Opium should not be given as a routine treatment. If the diarrhœa depend upon ulceration of the colon, large colon injections should be used. The hips being elevated, the injection should be allowed to flow in gradually from a fountain bag; in this way from two to four pints of injection are to be introduced, and it may be retained for some time. Simple water containing ʒj of soda or borax or salt to the pint may be used; the addition of an astringent is seldom, if ever, necessary. The stools should be examined to see what classes of food are undigested, and suitable dietetic regulations should be enforced. Meat, as a rule, is poorly borne. In all cases care should be taken to build up the general health and to avoid exposure to cold. A flannel abdominal bandage should be worn constantly. In some cases the best results are obtained by sending the patient to spend the winters in a warm, dry climate.

PHLEGMONOUS ENTERITIS.

This affection is exceedingly rare as a primary disease, but it may occur in connection with ulceration of the intestine, strangulated hernia, and intussusception. The purulent infiltration may be localized or diffused.

Symptoms.—The primary cases run a peracute course, with pain, tympanites, constipation, and fever, and terminate by septic peritonitis. These cases appear to be due to infection by the bacterium coli commune. The symptoms of the secondary cases may be obscured by those of the primary disease, so that the diagnosis is seldom made.

Prognosis.—The disease is rapidly fatal.

PSEUDO-MEMBRANOUS ENTERITIS.

Etiology and Synonyms.—Pseudo-membranous enteritis occurs (*a*) as a secondary process in acute infectious diseases, especially typhoid fever, scarlet fever, and cholera; (*b*) as a complication of dysentery or of intestinal obstruction; (*c*) in conditions of advanced cachexia; (*d*) as the result of poisoning by mercury, lead, and arsenic, and in uræmic conditions. *Synonyms:* Diphtheritic or Croupous enteritis.

Pathology.—The pseudo-membranous inflammation is usually more marked in the colon, but, especially in the mercurial and uræmic forms, the small intestine may also be involved. Necrosis, ulcerations, perforation, or hemorrhage may result, or cicatricial obstruction may ultimately develop.

The **symptoms** are latent in many cases secondary to acute infectious diseases and to cachectic states. In other cases, especially in the mercurial form, the symptoms of a violent entero-colitis are developed. Stools consisting of a thin, purulent liquid containing blood and shreds of the pseudo-membrane are fairly characteristic of this affection. Hemorrhage and perforative peritonitis are common complications.

This disease should not be confounded with membranous or mucous colitis—an entirely distinct disease.

The **treatment** is that of a severe acute entero-colitis. Opium should be given to limit peristalsis and to diminish the danger of perforation or hemorrhage.

MUCOUS COLITIS.

Etiology.—This affection regularly occurs in neurasthenic and hysterical patients. Over 80 per cent. of cases occur in women, especially in those who have suffered from uterine disease or from dyspepsia. It is essentially a disease of adult life, but a few cases occur in children. *Synonyms:* Mucous colic; Membranous colitis; Mucous diarrhœa.

Pathology.—There are no evidences of inflammation, but the disease seems to be due to a derangement of the mucous follicles of the colon. The exact nature of the disease is, however, unknown.

The **symptoms** appear in attacks characterized by severe colicky pain with tenderness over the abdomen, followed by the passage of mucus in flakes or strings or as casts of the lining of the bowel. Mucus is not, however, passed with every paroxysm. The attacks may last for several days or weeks; they may be produced by errors in diet or by mental worry, and they often occur in the early morning hours. The strings of mucus frighten the patients into the belief that the "lining of the bowel is ulcerated and is coming away," so that they become hysterical and hypochondriacal. Between the attacks the symptoms of neurasthenia are regularly present; they become more marked before and during the paroxysms.

The course of the disease is often chronic, the attacks recurring at intervals for years.

The **diagnosis** is easily made if the mucus be not mistaken for other substances, such as intestinal parasites and shreds of undigested food. The long continuance of the disease, the absence of fever, and the exciting rôle played by neurasthenia and the emotions serve to distinguish the disease from diseases of an organic basis.

The **prognosis** is good if the neurasthenia can be relieved.

Treatment consists primarily in the cure of the neurasthenia. The Weir Mitchell rest-cure should be tried in obstinate cases, while in all instances the nutrition should be improved in every way. Forced feeding, especially by cream and cod-liver oil, is often beneficial, and it can be said that the prognosis is good if the patient can be made to gain in weight.

Morphine is not to be used during a paroxysm, for fear of the habit being formed. High enemata of warm salt-solution (ʒj : Oj) may be given every day to cleanse the colon and to bring away the mucus. At least two quarts of the solution should be allowed to enter the bowel slowly, the patient lying on the left side with the hips elevated. Astringent and irritating enemata should not be used. Dietetic errors and digestive disturbances should receive appropriate treatment.

DIARRHŒAL DISEASES OF CHILDREN.

General Etiology.—Diarrhœal diseases occur with especial frequency among artificially fed children between the ages of six and eighteen months, and tenement-house and asylum children are more apt to be attacked. Owing to the small size of the child's stomach and to the deficiency in the saliva and in the proper acidity of the gastric secretions, dietetic errors result in graver consequences than in adults. The food may be given too freely or at irregular intervals, or the child may partake of food suitable only for adults, and the result of these dietetic errors is intensified by teething and by hot weather. Decomposed milk teeming with bacteria is perhaps the most common cause of infantile diarrhœa. The relation of bacteria to the diarrhœal affections of children has received careful attention. The healthy stools of children contain a number of micro-organisms, the most important of which are the bacterium coli commune and the bacterium lactis aërogenes, the latter being present only after a milk diet. These two bacteria are alone constantly present. In infantile diarrhœa there appear, in addition to the above-mentioned bacteria, a great number of micro-organisms, as many as forty varieties having been described. Acknowledging that these diarrhœal diseases have a bacterial origin, no one germ can be regarded as the specific cause, but a large number of different kinds are concerned.

Classification.—Three distinct forms of acute infantile diarrhœa are to be described: 1. Acute dyspeptic diarrhœa; 2. Acute entero-colitis; 3. Cholera infantum.

ACUTE DYSPEPTIC DIARRHŒA.

Acute dyspeptic diarrhœa, which is caused by the irritation of undigested or tainted food, is due to increased intestinal peristalsis.

Symptoms.—The stools, which are rarely more frequent than five or six in the twenty-four hours, consist of lumpy masses of undigested milk or food. They are not watery, and they contain no mucus. Their color is yellow, mixed with green, usually changing to green on exposure to the air. There may be vomiting of food and of mucus. Colicky pain usually precedes each stool, and the abdomen may be distended with gas. Convulsions or carpopedal spasms may occur in nervous children. In mild cases there may be no fever, but in the severer forms and in the case of children naturally feverish the temperature may reach 104° or even 105° F. The attack usually terminates when the intestine has been relieved of its irritating contents; it may, however, prove fatal in sickly children. In hot weather and in neglected cases the disease may develop into entero-colitis.

Treatment.—The bowels should be moved freely by castor oil or calomel, even if the condition of diarrhœa persist. Food should be withheld for a time until the stomach is settled, but cracked ice or cool water may be given. After the bowels have been moved by medication, bismuth and chalk mixture may be given.

ACUTE ENTERO-COLITIS.

Acute entero-colitis is the ordinary form of summer diarrhœa in children. It is the dreaded scourge of tenement-house children in their second summer, and it often appears as a sequel to the specific diseases of children. The ileum and the colon are the seat of a catarrhal inflammation, the follicles being especially involved, and frequently ulcerated, so that the name "follicular enteritis" or "follicular dysentery" is sometimes applied to these cases. In severe cases the inflammation may be of the pseudo-membranous variety.

Symptoms.—The disease usually follows acute dyspeptic diarrhœa. The general condition of the child becomes worse. The temperature rises and remains constantly high, although extreme hyperpyrexia is but seldom observed. The stools become small in size, vary between twelve and thirty in the twenty-four hours, contain a large quantity of mucus frequently mixed with blood, and are acid and offensive. They may be passed painlessly, or with straining and tenesmus if the rectum be involved, or colicky pain may precede the movement. Flatus is usually passed in considerable amount, and the abdomen is distended, hard, and usually tender along the line of the colon. Vomiting may occur, but it is rarely a marked feature of the disease. The attack may last for several weeks and may terminate by recovery or by death from inanition and exhaustion, or the affection may become protracted. In this form the acute symptoms disappear, but the diarrhœa continues and nutrition does not improve. From this subacute enteritis the child may convalesce in from six to eight weeks, or the disease may become chronic.

In some cases of entero-colitis the follicular glands of the colon are extensively involved, leading to the symptoms frequently spoken of as the "acute dysentery of children." The onset is sudden, with fever, convulsions in the severer forms, and frequent small passages almost entirely composed of mucus and blood, and not containing feces. There are incessant abdominal pain and rectal tenesmus. In mild cases the bowels can be opened within two or three days by castor oil, and recovery rapidly ensues; but if the bowels cannot thus be moved, the case is apt to continue from three to six weeks, or even to become chronic.

There is a rare form of entero-colitis, of great severity, characterized by high fever, dysenteric symptoms, convulsions, stupor, and collapse.

Treatment.—Attention to the proper feeding of the child is of the utmost importance. The diet should consist of sterilized or peptonized milk or of albumin-water. Cream and water are often better borne than milk. After the irritating cause has been removed, bismuth and chalk mixture should be given, to which any of the intestinal antiseptics may be added. Of these, bismuth salicylate, bismuth subgallate, and salol are perhaps the most reliable. Large enemata of salt-solution (ʒj : Oj) should be used to flush out the colon; they are of the greatest service. The addition of astringents to these enemata is not to be recommended. Should dysenteric symptoms occur, the bowels should be opened with castor oil, and this medication should be repeated every other day. Medicated enemata may in these cases be used, as in the dysentery of adults.

CHOLERA INFANTUM.

The term "cholera infantum" has been applied loosely to any diarrhœa of childhood with vomiting and prostration, but it should be limited to a special group of symptoms resembling cholera morbus of the adult. The term being limited in this way, cholera infantum is rather a rare disease, occurring in only 2 or 3 per cent. of the summer diarrhœas of children. Cholera infantum is essentially a bacterial disease, the symptoms being due to poisoning by the ptomaïnes generated by the action of the bacteria upon milk or other intestinal contents, although no one organism can be described as a specific cause.

Pathology.—There is an acute gastro-enteritis, although the appearances of inflammation may disappear after death, leaving the mucous membranes pale and sodden. The lymph-glands of the intestine may be swollen, and in rare instances may soften and break down to form follicular ulcers. Various micro-organisms may be found in the mesenteric glands and in the lymphatic vessels of the intestinal wall.

The **symptoms** of cholera infantum may begin abruptly, or there may be a preliminary diarrhœa for several days. The first regular symptom is purging. The stools, at first acid and fecal, later become alkaline, serous, and either colorless, brownish, or of the "rice-water" appearance. The passages are abundant, often are expelled with force, and in some cases amount to a constant discharge. The purging is supposed to result from paresis of the blood-vessels of the intestinal wall from the toxic action of the ptomaïnes, resulting in the abundant transudation of serum. There is, as a rule, neither abdominal pain nor tenderness, although in some cases colicky pain may precede the purging in the earlier stages of the disease. Vomiting is not as constant as purging, but it may be violent and incessant, so that no food can be retained. The vomited matters consist first of food; later they are of a brownish or bile-stained serum, and may be like rice-water. The skin is cool and clammy, but the rectal temperature shows an increase to 103°, 105°, or even 107° F., there being no disease of infancy regularly attended by so high a temperature. The pulse is rapid and thready. The skin may be firm and hard—a condition to which the names "sclerema" and "frozen skin" have been applied. The appearance of the child is rapidly altered; the face is thin, drawn, and of an ashy paleness, the eyes are sunken, the fontanelle is depressed, and the loss of weight is evident. The urine becomes diminished or even suppressed, and uræmic symptoms may develop.

Course of the Disease.—1. A considerable number of the children die in from one to three days, from exhaustion, collapse, or with cerebral symptoms. These "hydrocephaloid" symptoms, or "pseudo-hydrocephalus," consist of drowsiness merging into coma, muscular twitchings or convulsions, retraction of the head, and subnormal temperature or hyperpyrexia; the respirations become shallow and irregular, and may be of the Cheyne-Stokes variety. The pulse becomes irregular and flickering. The vomiting and purging usually cease for some hours preceding the fatal termination.

2. Other patients begin to improve in from twenty-four to thirty-six hours, and the improvement is either rapid and complete or is complicated by a recurrence of the former symptoms. Chronic furunculosis may appear as a sequel.

3. In some children improvement progresses only to a certain point, but the child still continues sick, with vomiting, diarrhœa, and prostration. In this condition the child may remain for weeks and then slowly recover; or the symptoms may continue, prostration and emaciation may become more and more marked, and the child may ultimately die from marasmus.

The **prognosis** is always serious, especially in bottle-fed babies and in asylum and tenement-house children. A guarded prognosis must always be given, however mild the case may appear at the onset.

Treatment.—The most important indication for treatment is the reduction of the hyperpyrexia. This should be accomplished by baths of 90° F. gradually reduced to 80° F. by the addition of cool water. Irrigation of the stomach and the colon is indicated in every case, to remove toxic products and to supply water to the tissues. For the purging, opium is almost indispensable, but the drug should be given to children with extreme caution, as they are peculiarly sensitive to its action. Morphine gr. $\frac{1}{100}$ is a fairly large dose for a child of one year. As a practical rule, opium should not be given to babies under six months of age unless it be absolutely necessary to do so.

Intestinal antiseptics should be given if they do not add to the vomiting. Of these, bismuth salicylate (gr. v q. 2 h.) is perhaps the most useful. Small doses of mercurial preparations often are of service in controlling the vomiting. Calomel (gr. $\frac{1}{12}$), mercury with chalk (gr. $\frac{1}{12}$), or bichloride of mercury (gr. $\frac{1}{200}$) may be given in these doses every two or three hours without danger of salivation. Stimulants may be given if indicated, and, should collapse appear, subcutaneous injections of a 1 per cent. saline solution (sterilized) may be given as in Asiatic cholera. Iced drinks may be given even if they are not retained. If the case be protracted, it is of the utmost importance to move the child to the country, where the air is cool and fresh. These little patients stand travel well, and the improvement is often striking within a few hours after the change has been made. The dietetic rules are those applicable to acute entero-colitis.

Cholera infantum in meat-fed children presents certain peculiarities by which it differs from cholera infantum of milk-fed babies. The stools are not as watery, as frequent, nor as profuse as in the ordinary cases, but are grayish or yellowish-green and highly offensive. Tympanites is a constant symptom, but vomiting is more frequently absent. The general symptoms are those of sepsis, and death usually results in from forty-eight to seventy-two hours. Mild cases may recover.

CHOLERA MORBUS.

Etiology and Synonym.—Cholera morbus is most common in young adults and in the summer months. The attack may be induced by improper or partially decayed food, by unripe fruit, or by impure drinking-water. At times the disease assumes such epidemic proportions that it seems as though some specific micro-organism must act as the exciting cause. *Synonym:* Cholera nostra.

Pathology.—The lesion consists of a catarrhal inflammation of the stomach and of the large and small intestines. The submucosa is infiltrated to some extent by fibrin, serum, and round cells, and the intestinal glands are swollen. It is impossible to differentiate cholera morbus from Asiatic cholera except by post-mortem examination, the comma bacillus being found in the latter disease.

Symptoms.—The attack is usually sudden, although it may be preceded by oppression and by vague abdominal distress. The patient is usually attacked during the night or the early morning. There are nausea and faintness with violent and incessant vomiting, the ejected matter consisting at first of food and later of fluid, either colorless or tinged with bile. Following or coincident with the vomiting is severe purging. The stools at first are feculent, but later become watery and odorless; they consist of serum with flakes of desquamated epithelium, giving to the stools the so-called "rice-water" appearance. The color of the passages is usually greenish or yellowish, and the fluid is sufficiently acrid to irritate and excoriate the anal parts. Cramps in the abdomen cause tearing, lacerating pain, usually referred to the umbilicus, but in some cases the purging is painless. There may be violent muscular cramps, especially in the calves of the legs, due to the dryness of the tissues. Prostration appears early in the attack. The face is drawn and anxious; the pulse is rapid and thready; the skin is cold and clammy, but the internal temperature reaches 101° or 102° F., or even higher than this.

The **duration** of the attack varies from a few hours to several days.

The **prognosis** is good. The disease may, however, be fatal in debilitated subjects, so that the whole mortality is between 2 and 3 per cent.

The **diagnosis** from Asiatic cholera cannot be made with certainty without bacterial examination of the stools.

Treatment is entirely symptomatic. As drugs cannot be retained when given by the mouth or the rectum, hypodermic medication alone is to be relied upon. Morphine in gr. ¼ doses should be given, and repeated if necessary, to check the pain, vomiting, and purging. Hot poultices applied to the abdomen afford relief. For the attendant thirst cracked ice may be allowed if it does not increase the vomiting. It is well to give no food by the mouth until the attack is well over, and then food may be allowed in small quantities at first and at frequent intervals.

DYSENTERY.

Etiology.—Dysentery is essentially a disease of the tropics, where "it destroys more lives than cholera, and it has been more fatal to armies than powder and shot" (Osler). Under imperfect hygiene sporadic and endemic cases occur in Northern cities, and even epidemics may occur, but with improved sanitation the disease is much less common than formerly. The exciting cause has been supposed to be drinking-water contaminated by animal matter, while in the tropics one form of dysentery appears to be due to infection by the amœba coli. Dysentery attacks patients of all ages, and there is no racial exemption. The majority of cases occur in the late summer and early fall months.

Varieties.—The following varieties are to be described: 1. Acute catarrhal dysentery; 2. Amœbic or tropical dysentery; 3. Acute croupous dysentery; 4. Chronic dysentery.

ACUTE CATARRHAL DYSENTERY.

This orm is the one usually seen in temperate climates.

Pathology.—The inflammation is limited to the large intestine, especially to the sigmoid flexure and the rectum, but the lower portion of the ileum may also be involved. The mucous membrane is swollen, congested, and covered with tenacious blood-stained mucus. The solitary glands are prominently enlarged, and in severe cases may ulcerate. In the gravest form of the disease the follicular ulceration may deepen and spread, more extensive ulcerations being formed. In children the inflammation and ulceration of the follicles are more marked than in adults, so that the disease is often spoken of as "acute follicular dysentery."

The **symptoms** differ according to whether the rectum or the upper colon be involved.

I. If the rectum be involved, as it is in the vast majority of cases, there may be a preliminary diarrhœa with griping pains; in other cases the disease begins abruptly. An initial chill is rare. Diarrhœa is usually the first symptom. At first fecal and painless, the stools change their character within from twenty-four to thirty-six hours, becoming characteristic of the disease. The dysenteric stools are first composed of mucus with a few scybalous fecal masses, but finally they consist only of mucus and blood ("bloody slime"). The presence of pus in the stools indicates follicular ulceration. The stools are frequent, varying from ten to two hundred in the twenty-four hours, and are small in quantity, rarely exceeding half an ounce. They are passed with a straining, bearing-down pain referred to the rectum. This tenesmus is more marked during and after a movement, but it may be more or less continuous, amounting to a constant desire to go to stool, and the patient may complain of burning pains referred to the rectum. Preceding each stool there is apt to be severe colicky pain in the abdomen, with possibly some tenderness along the descending colon. The temperature is not high, varying from 101° to 103° F. at the outset. There are apt to be nausea and vomiting with incessant thirst. Prostration may be extreme. Strangury may attend the rectal tenesmus, and in severe cases the urine may contain albumin and casts. As the patient improves the number of passages diminishes, the mucus becomes opaque and less discolored by blood, and fecal matter begins to be passed. Rectal tenesmus finally disappears.

2. If the inflammation involve the upper colon, and not the rectum, the clinical picture is different. The patient passes not mucus and blood, but large, watery, feculent stools without tenesmus. In severe cases blood may be admixed with the stools. Colicky abdominal pain precedes each passage. The constitutional symptoms are not severe, and the prognosis is that of the first form. In children this form is accompanied by extreme prostration and febrile disturbance, so that the case may be mistaken for typhoid fever.

The **prognosis** for the attack is good except in the extremes of life. If the bowels can be opened within two days by castor oil, the attack will probably not be severe. It is possible for the disease to run into a chronic form. Peritonitis and abscess of the liver rarely occur.

Diagnosis.—In children the disease may be mistaken for intussusception, while in adults cases of fecal impaction or of cancer of the intestine are frequently treated as dysentery

TROPICAL OR AMŒBIC DYSENTERY.

Pathology.—The amœba coli or amœba dysenteriæ, a one-celled protoplasmic organism showing active amœboid motion, is from 10 to 20 micromillimeters in diameter. It has been proven to be the inciter of dysentery in tropical and sub-tropical countries, and it is not uncommonly found in the Northern United States and in Europe. It probably gains access to the body by the medium of drinking-water. The large intestine is involved, particularly the descending colon, but the lower part of the ileum may also be affected. The lesion consists of œdema of the mucosa, with localized areas of cellular infiltration causing little elevations upon its surface. These elevations become necrotic and are cast off, exposing a yellowish-gray gelatinous mass which subsequently sloughs, leaving an ulcer with infiltrated and undermined edges extending through the submucosa and even to the serous layer of the colon. Extensive undermining of the edges of the ulcerations allows of the formation of fistulous tracts bridged over by apparently healthy mucous membrane. The colon may be so involved that the remaining mucous membrane projects like little islands from the surrounding ulcerations. The disease extends by progressive infiltration and ulceration of the mucous coat of the intestine, and in severe cases large areas may slough and be thrown off *en masse.* A croupous inflammation of the colon complicates amœbic dysentery in some cases. The microscope shows a notable absence of the products of purulent inflammation, but reveals amœbæ in large numbers in the floors and the walls of the ulcers. Healing of the ulcerations by cicatrization may lead to subsequent stricture of the intestine. One-fifth of the cases are complicated by lesions in the liver. There may be areas of necrosis of the parenchyma of the liver, or there may be single or multiple abscesses, consisting of necrotic liver-tissue with a small amount of pus. Amœbæ are constantly found in the contents of the abscesses. Rupture of an abscess of the liver into the right pleura or lung is not infrequent.

Symptoms.—The onset of the disease is usually gradual, beginning as a diarrhœa; in severe cases, however, the disease may begin abruptly. The temperature is but moderately raised, if at all. Pain and tenesmus may be present at the outset of severe cases, but they are not constant throughout the disease. The principal symptoms are diarrhœa and a progressive loss of flesh and of strength. At the onset the stools may be mucoid and blood-stained as in catarrhal dysentery, but the characteristic stools are fluid, contain mucus and possibly blood, are of a yellowish-gray color, and in them are to be found actively moving amœbæ. The number of the passages varies from six to twelve in the twenty-four hours. The diarrhœa runs an irregular course of from four to eight weeks, with periods of temporary improvement.

Prognosis.—Recovery is usually slow and tedious from anæmia and muscular weakness, and convalescence may be interrupted by relapses. The disease has a greater mortality than catarrhal dysentery, and shows a tendency to become chronic. Death may result from the severity of the local inflammation, from exhaustion in the prolonged cases, or by reason of the liver complications.

ACUTE CROUPOUS DYSENTERY.

This form of dysentery may occur in a primary form or may appear as a terminal complication of chronic heart disease, of chronic Bright's disease, of profound cachectic states, or of lobar pneumonia.

Pathology.—The colon is thickened and infiltrated by fibrin, serum, and pus-cells, so that the mucosa, from the ileo-cæcal valve to the rectum, appears as a yellowish exudate without trace of glandular structures. In mild cases the tops of the folds of the colon are covered with a thin yellowish or grayish pellicle. The mucosa thus infiltrated undergoes necrosis, and sloughs, leaving large irregular ulcers which may involve the submucosa and even the muscular wall. Perforation of the ulcers is not uncommon. In some cases the lower portions of the colon alone are involved. Peritonitis may complicate the disease even without actual rupture, and ulcer of the liver may result from infective thrombosis of one of the mesenteric veins. Should the patient recover, the ulcerations will cicatrize and stricture of the intestine may result; but the healing of the ulcers is very slow, and in many cases chronic ulcerations are left.

The **symptoms** of acute croupous dysentery resemble those of the catarrhal form, but are more severe. If the rectum be involved, tormina and tenesmus are extreme, the stools are numerous, are composed of mucus, blood, pus, and shreds of sloughing membrane, the temperature is high, prostration is extreme, and the patient is seriously ill from the onset. If the rectum be not involved, there is no tenesmus; the stools are diarrhœal in character, contain blood, pus, and shreds of membrane, and are accompanied by severe abdominal pain. The constitutional symptoms are severe, closely resembling those of typhoid fever.

In the secondary cases the patient is already ill from the primary disease, so that the course of the dysentery is insidious. There is usually a moderate diarrhœa, with the occasional passage of blood and mucus.

The **prognosis** of croupous dysentery is exceedingly grave. Death may result from the severity of the inflammation, from peritonitis or perforation, or from abscess of the liver; or the patient may pass into the typhoid condition or may die exhausted. Should the patient recover from the acute attack, the condition may become chronic.

Complications.—The course of the disease may be complicated by septic arthritis, endocarditis, pericarditis, pleurisy, and pyæmia. Peripheral neuritis with paraplegia is a rare complication. The occurrence of pylephlebitis and abscesses in the liver has already been alluded to. Chronic nephritis has in some cases followed the disease.

Treatment of Acute Dysentery.

Of the Catarrhal Form.—The patient should be put to bed, no matter how trifling the attack may seem, and should be kept on a milk or liquid diet. It is important that the bowels should be moved at the beginning of the attack, castor oil being the preferable laxative. An estimate of the probable duration can often be made with reasonable accuracy by the promptness of the response to the laxative remedies. The bowels should be opened every second day, if possible. Opium should be given by the mouth or by suppository, to control the tormina and the tenesmus. Much relief is often afforded by rectal medication. An enema of hot starch-water containing from 10 to 15 drops of laudanum may be given after every passage. As the enemata are seldom retained for any length of time, poisoning is not likely to occur. Suppositories containing $\frac{1}{8}$ grain of cocaine hydrochlorate, or injections of from 5 to 10 drops of a 4 per cent. solution of the drug, are serviceable in mitigating the pain, but the absorptive power of the rectum is so great that toxic symptoms may occur unless the case is watched with care. Quinine (gr. x–xv daily) should be given if there be a malarial history. Bichloride of mercury (gr. $\frac{1}{100}$) may be given every two hours, and large doses of bismuth subnitrate (ʒj q. 2 h.) are often of great service. Astringent rectal injections are not recommended in acute cases.

In severe cases ipecacuanha may be given in large doses. This drug, without doubt, is of great service, especially in the cases occurring in the tropics. A hypodermic injection of morphine should be given, followed in half an hour by from 20 to 40 grains of powdered ipecacuanha in capsule. A second injection of morphine should be given if vomiting threaten. No food should be given for six hours after the dosage. If vomiting occur, the dose may be repeated within a few hours.

Amœbic Dysentery.—Besides the above-mentioned treatment, colon-irrigation of quinine (1 : 2500) may be used, the amœbæ being rapidly killed by the drug. Medication should be continued in these cases until the amœbæ are no longer present in the stools.

For *croupous dysentery* the treatment is that of the catarrhal form, except that opium is required in larger doses, and stimulants are needed to support the strength of the patient. Should extensive ulceration exist, purgatives must be administered with caution.

CHRONIC DYSENTERY.

Etiology.—Chronic dysentery may be chronic from the first or may succeed an acute attack. Many cases of amœbic dysentery may be subacute from the start.

Pathology.—The mucosa is thickened, pigmented, and presents an irregular puckered surface. The submucosa and the muscular coats are usually hypertrophied, and the lumen of the colon may be diminished. Cystic degeneration of the glandular structures may be visible to the naked eye. Ulcerations are generally present in all stages of development; they are usually pigmented.

Symptoms.—Tormina and tenesmus are uncommon unless during acute exacerbations. The stools vary from three to fifteen in the twenty-four hours, and are not always of the same character; they may be composed of mucus, occasionally stained with blood, or they may be liquid and frothy, consisting of feces, mucus, and undigested food.

Blood and shreds of tissue are not common except during the acute exacerbations. There may be constipation alternating with diarrhœa. From time to time will appear acute exacerbations in which the stools assume a more characteristic appearance of dysentery and are accompanied by tormina and tenesmus. Pain and tenderness along the colon are usually present. The patient rapidly loses flesh and strength; the tongue resembles raw beef; digestion is interfered with; flatulence is common; anæmia and emaciation reach finally an extreme degree.

Prognosis.—The course of chronic dysentery is prolonged for months or possibly for years, the patient usually dying from exhaustion and inanition.

Treatment.—Proper diet is of prime importance. The patient should be put to bed and be given a milk diet. Should curds appear in the stools, meat broths or scraped beef may be given, and only such food be allowed as may be digested thoroughly. Inspection of the stools should then govern the diet. Colon-irrigation should be employed daily, simple salt-solution (ʒj : Oj) being the preferable solution. Medicated and astringent injections are painful, and are not more efficacious than simple cleansing solutions.

APPENDICITIS.

Etiology.—Appendicitis is more common in the young than in the old, one-half the cases occurring before the twentieth year. Males are affected in three-fourths of the cases. In a little less than half the cases hard fecal concretions or foreign bodies, such as grape-seeds, orange-pips, etc., are found in the diseased appendix; on this point, however, statistics are misleading, the percentage of cases in which concretions are found being estimated as low as 6 per cent. by some observers, while the concretions are said to be present in 10 per cent. of presumably healthy appendices. The question as to whether foreign bodies and concretions can originate an attack of appendicitis is still unanswered. It would seem, however, that foreign bodies, concretions, exposure to wet and cold, injury and overstraining, and previous inflammations of the appendix or the narrowing of its lumen by stricture or by twists, render the appendix more susceptible to the infection of micro-organisms, so that they may be considered as causes predisposing to a bacterial infection.

ACUTE CATARRHAL APPENDICITIS.

Pathology.—The mucous membrane lining the appendix is in a condition of catarrhal inflammation; the walls are swollen and are infiltrated by serum and leucocytes, so that the appendix is enlarged and its lumen becomes contracted, especially toward the cæcal end. The peritoneum covering the appendix is congested, is coated with fibrin, and is adherent to neighboring peritoneal surfaces. In this, the mildest and commonest form of appendicitis, there is no general peritonitis, no abscess, and no perforation.

Symptoms may begin gradually or suddenly. If gradually, there is a preliminary diarrhœa, or diarrhœa alternating with constipation, and a pain which is either of a colicky character or is localized in the right iliac fossa. Cases with a sudden onset are initiated by a chill or by chilly feelings. When the disease has developed there is fever, usually of moderate intensity, running between 101° and 103° F., remaining for three or four days and then slowly subsiding. It is of the greatest importance to keep accurate records of the temperature of each patient,

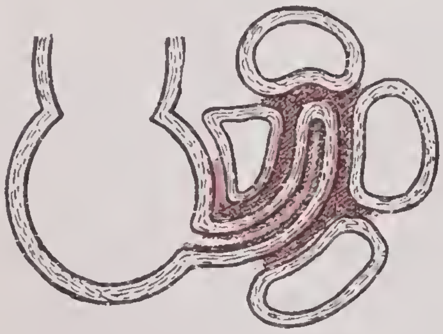

FIG. 43.—Catarrhal appendicitis: localized fibrinous peritonitis; no pus-formation; no general peritonitis.

as it is impossible at the onset to distinguish between the mild and the severe forms, and the exact diagnosis, the presence of complications, and the indications for operative interference are all determined, in great measure, by the character of the temperature curve. With the fever there are headache, loss of appetite, nausea and vomiting, and prostration.

Local symptoms consist of pain, tenderness, and position in bed.

Pain, which is localized in the right iliac fossa, may be steady or paroxysmal. Should the appendix be abnormally situated, the pain may be felt in the right lumbar region or nearer the median line in front. In some cases the pain cannot accurately be localized.

Tenderness is usually elicited by firm continuous pressure at McBurney's point, situated from one and a half to two inches from the right anterior superior spine, on a line drawn between this bony prominence and the umbilicus. If the appendix be displaced behind the cæcum, tenderness may not be elicited at McBurney's point, but may be detected by vaginal or rectal examination ; hence, in doubtful cases, these methods of examination should always be resorted to. The thigh is usually flexed to relax the anterior abdomen; it may be adducted from irritation of the obturator nerve within the pelvis.

Physical Examination.—Tenderness is produced by pressure at McBurney's point or by rectal or vaginal examination. There is usually considerable rigidity of the abdominal wall in the right iliac region. There may be a feeling of resistance in the right iliac region, and slight dulness on percussion, but no defined tumor can be appreciated. The detection of resistance and dulness on percussion depend upon the position of the vermiform appendix (not being appreciable should the appendix be behind the cæcum) and upon the extent of the localized peritonitis. Should several coils of intestines be matted together and adherent to the appendix, the resistance and dulness may be as well marked as in cases of the suppurative form.

Course of the Disease.—After from two to four days the patient begins to improve, showing that the case is one of the mild form, and in about a week convalescence is thoroughly established. Pain, tenderness, and irregular action of the bowels may, however, persist for some weeks, owing to the resulting peritoneal adhesions, and relapses may occur at any time.

ACUTE SUPPURATIVE APPENDICITIS (ULCERATIVE APPENDICITIS).

Pathology.—In this form of appendicitis the wall of the appendix is infiltrated by fibrin, serum, and pus, and in severe cases the wall may slough in some part, so that the contents of the appendix escape into the peritoneum. The adjacent peritoneal surfaces are inflamed, coated with fibrin and pus, and become adherent, so that there is formed a cavity containing pus. This circumscribed intraperitoneal abscess may remain localized or may rupture into the general peritoneal cavity, producing acute peritoneal septicæmia; or diffuse suppurative peritonitis may result without actual rupture. Suppuration may extend along the connective tissue in the mesentery of the appendix and invade the retroperitoneal tissues. This formation of an extraperitoneal abscess is not common, and occurs only in connection with the intraperitoneal abscess

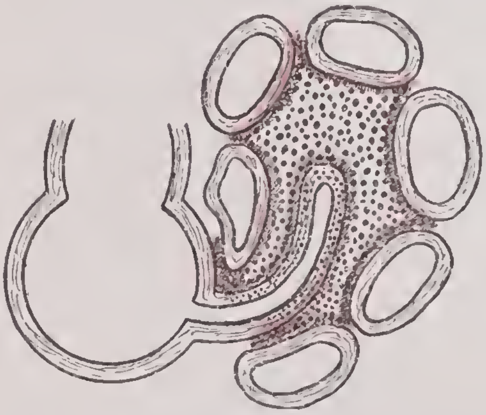

FIG. 44.—Suppurative appendicitis: limiting adhesions; localized intraperitoneal abscess.

previously described (Figs. 44, 45). The peritoneum dividing the intraperitoneal from the extraperitoneal abscess ultimately becomes absorbed, so that one large abscess-cavity results.

The pus may be abundant and creamy, or the quantity may be small and surrounded by a large amount of inflammatory tissue. The pus is usually grayish in color, exceedingly offensive, and, should sloughing of the vermiform appendix occur, may be admixed with the contents of the intestine. Bacterial examination usually reveals pure cultures of the bacillus coli commune.

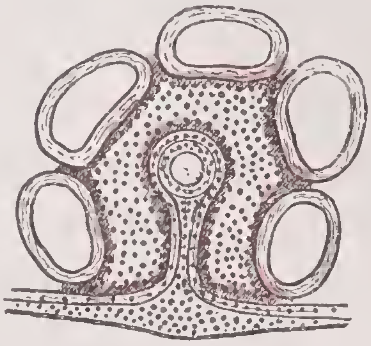
FIG. 45.—Cross-section of appendix with suppurative inflammation, showing extension of the infection along the connective tissue of the mesentery to the retroperitoneal connective tissue, and the formation of an extraperitoneal abscess. The peritoneum separating the intraperitoneal from the extraperitoneal suppuration is here shown intact; it, however, subsequently disappears.

The position of the abscess varies according to the position of the appendix. The usual situation is in the angle of the ileum and the cæcum, lying on the psoas muscle. In other cases the abscess lies behind the cæcum or extends into the pelvis.

Rupture of the abscess may occur at any time, either into the peritoneum, the intestine, or the bladder, or, less frequently, through the abdominal wall, the pleura, the portal vein, or the iliac artery. When the retroperitoneal tissues become infected, the so-called "perityphlitic" abscess may burrow under the iliac fascia and appear below Poupart's ligament, or may extend to the perinephritic tissues. Suppuration may extend along the psoas fascia or may involve the perirectal connective tissue. Burrowing through the obturator foramen, the pus may appear as a gluteal abscess.

The **symptoms** begin like those of the catarrhal form, but they are more severe. The temperature ranges between 102° and 104° F.; pain and tenderness are well marked; the thigh is flexed and adducted; the bladder and the rectum may give evidences of irritability.

The **physical signs** during the earlier stages of the disease depend upon the position of the abscess.

(*a*) If the abscess be in the inner side or in front of the cæcum, there will be tenderness in the right iliac fossa, especially marked over McBurney's point. There may be some bulging in the right iliac fossa, in which region a percussion-note of dulness is obtained. The abdominal wall on the right side is rigid, and there is an indistinct tender mass to be felt in the region of the appendix.

(*b*) If the abscess be small and be situated behind the cæcum and the distended intestines, tenderness on palpation will be elicited, but no tumor can be detected, although there may be a sense of resistance to palpation. The percussion-note is tympanitic, and some œdema may be noticed in the right lumbar region.

(*c*) If the abscess encroach upon the pelvic space, the physical signs may not be elicited by external abdominal examination, but rectal or vaginal examination detects the presence of the abscess.

Instead of the disease subsiding in three or four days, as does the catarrhal form, the symptoms become aggravated. The temperature becomes irregular and remittent, and septic symptoms appear on the third or fourth day and are strikingly developed by the end of the first week. In some cases there are added the symptoms of intestinal obstruction from septic paralysis of the intestinal wall.

Physical examination by the seventh or tenth day may reveal, by palpation through the abdominal wall, the rectum, or the vagina, an indistinct wave of fluctuation in the tumor-mass. The fluctuation is most marked in extraperitoneal abscesses, and is detected above Poupart's ligament or above the crest of the ilium. The abscess may, however, be small, and may be in a locality where it cannot be felt, so the diagnosis may still be doubtful. The aspirating-needle should never be used for diagnostic purposes unless a marked tumor with dulness is present in the cæcal region, and even then it should be used with extreme caution.

Course of the Disease.—1. Some patients gradually recover. The temperature declines, pain and tenderness subside, and the physical signs clear up. These are the cases of mild infection in which the peritoneal exudate is chiefly fibrino-serous without much admixture of pus, so that absorption of the effusion is possible.

2. Other cases go on with the symptoms of sepsis and of a localized peritoneal abscess.

(*a*) Some cases are operated upon and the pus is evacuated.

(*b*) In some cases the abscess ruptures. If the rupture occur through the abdominal wall or into the bladder, the rectum, or the intestine, the pus will appear externally, the temperature will fall, and the general symptoms will improve. If drainage be good, a spontaneous cure will result, but if drainage be poor, the abscess-cavity will fill up again and the old symptoms will reappear. Fistulæ may result, and there may be a resulting cystitis which, if the bladder be perforated, may prove fatal.

(*c*) Burrowing may occur, so that pus will rupture into distant parts, even into the pleural cavity.

(*d*) Acute progressive peritonitis may develop. This is the greatest danger, and usually begins in the second, third, or fourth day, before the limiting adhesions have become firm enough to prevent general infection of the peritoneum. Spreading abdominal pain, tympanites, and an increase in all the constitutional symptoms indicate the onset of the peritonitis. In some cases there is a progressive increase in the size of the tumor.

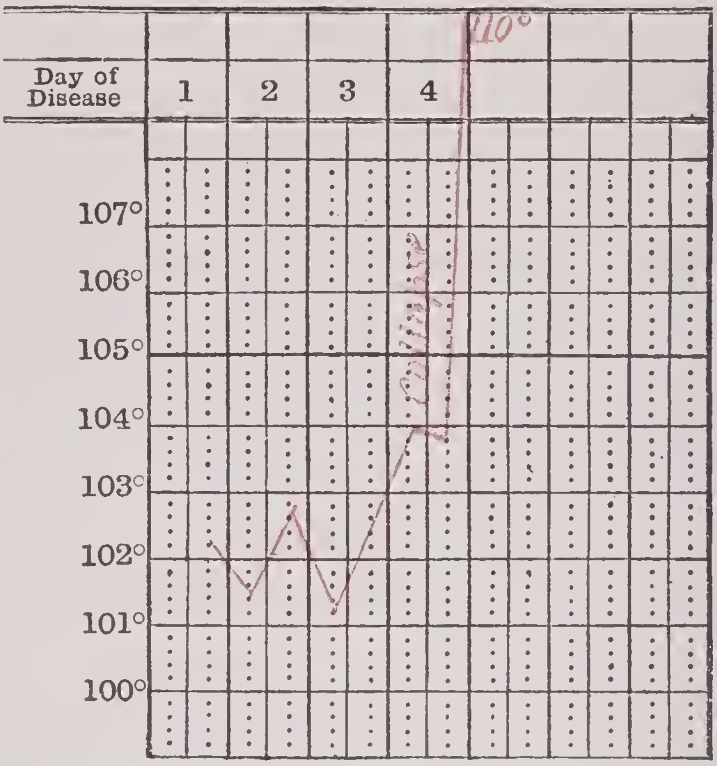

Fig. 46.—Suppurative appendicitis: rupture of abscess into peritoneal cavity; acute peritoneal sepsis.

(*e*) The abscess may rupture and discharge its contents into the peritoneal cavity, so that acute peritoneal sepsis may result. The temperature falls, but subsequently rises to a higher point than before, and collapse symptoms appear. Death may occur in from twelve to fifteen hours from collapse, with high ante-mortem temperature, or the symptoms of a general peritonitis will develop from which the patient will die in two or three days (see Fig. 46).

3. The course of the disease may be modified by complications. Among these may be mentioned thrombosis of the femoral vein, and thrombosis of the portal vein which may be infective, leading to pyæmia with multiple abscesses in the liver. Pyæmia or septicæmia may develop in neglected cases.

GANGRENOUS APPENDICITIS.

Under the above heading are included the rather rare cases in which primary gangrene or necrosis of the appendix-wall occurs, so that the contents of the appendix are discharged into the peritoneal cavity before there is time for limiting peritoneal adhesions to be formed. The patient usually has suffered from previous attacks of catarrhal or suppurative appendicitis, with resulting adhesions, so that the appendix becomes distorted and twisted. Usually the exciting cause is a foreign body or a fecal concretion which enters the appendix and induces a pressure-necrosis on its wall. There may be a local peritonitis, but no adhesions are formed, so that the sloughing appendix lies free in the peritoneal cavity. Acute peritoneal sepsis develops, and usually runs a fatal course.

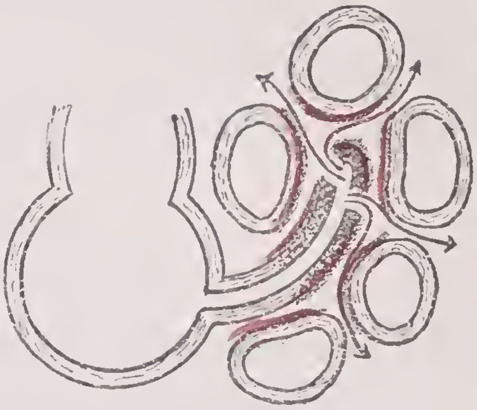

FIG. 47.—Gangrenous appendicitis; sloughing of the wall; escape of contents into peritoneal cavity; absence of limiting adhesions.

The **symptoms** begin like those of the suppurative form, but the patient is evidently more sick. Peritoneal sepsis and death usually result before a tumor can be appreciated.

Prognosis of Appendicitis.

In the *catarrhal form* the prognosis is good. It should be remembered, however, that the appendix is left adherent and predisposed to recurrent attacks.

In the *suppurative form* the prognosis is exceedingly good, depending less, however, upon the intensity of the inflammation than upon the skill of the surgeon. When general peritonitis occurs the prognosis is most unfavorable. Rupture of the abscess into the general peritoneal cavity is almost regularly fatal. Spontaneous cure may occur by absorption or by perforation through the abdominal wall or into the bladder or the rectum, but aside from these cases the prognosis is bad unless the case be treated surgically and the abscess-cavity be drained.

Gangrenous appendicitis, unless operated upon very early in the course of the disease, before actual perforation has occurred, is rapidly fatal.

Treatment of Appendicitis.

No disease requires more judgment for its proper treatment than appendicitis, as no routine plan of treatment is applicable to all patients. In every case a surgeon should be called into consultation, as the disease is, properly speaking, a surgical one. The treatment is both medical and surgical.

Medical Treatment.—The patient should be put to bed, no matter how mild the case may appear, and be put upon a liquid diet. Opium is to be given, to quiet the patient and to allay pain, but large doses to the point of semi-narcotization are not to be recommended. Cathartics are absolutely contraindicated, because of the danger of rupturing limiting peritoneal adhesions by the increase of intestinal peristalsis. The bowels should, however, be moved every second day by enemata of tepid water containing salt (ʒj : Oj). An ice-bag should be applied to the cæcal region, or a Leiter cold coil may be used. Hot poultices are not recommended.

Surgical treatment consists in evacuation of the pus, removal of the diseased appendix, and drainage of the abscess-cavity.

Operative interference is indicated under the following conditions: (1) In catarrhal appendicitis with severe constitutional symptoms, especially if the attack be recurrent;

(2) whenever symptoms of pus-absorption are present, whether the tumor can be felt or not; (3) whenever the patient is more than ordinarily sick, and does not seem to be standing the disease well; (4) should septicæmia develop; (5) in cases of spreading peritonitis; (6) in case of rupture and peritoneal sepsis; (7) in case of burrowing of the abscess; (8) in all cases in which a tumor presents itself; (9) in case of rupture into the bladder, the intestine, or the rectum, the drainage being imperfect.

CHRONIC APPENDICITIS.

This form of appendicitis occurs in patients who have had previous attacks of acute inflammation of the appendix.

Pathology.—The appendix is usually enlarged and of a sausage shape. Its walls are thickened, the outlet is stenosed, and its cavity is filled with mucus. The appendix may be sharply bent upon itself or displaced. The peritoneum adjacent to the appendix is thickened and adherent. There may be collections of serum or of sero-pus encapsulated by the peritoneal adhesions.

Symptoms.—In some cases there are recurring attacks of acute appendicitis, in the intervals of which the patient is perfectly free from trouble. To these cases the name "recurring appendicitis" is frequently applied.

In other cases the patient suffers more or less in the intervals from the chronic inflammation of the appendix; to these cases the terms "chronic appendicitis" and "chronic relapsing appendicitis" are more properly applied.

The *symptoms between acute attacks* consist of localized pain and tenderness, disturbances of digestion, and irregular action of the bowels. There is usually progressive loss of flesh and strength. Physical examination usually reveals tenderness over McBurney's point, a tumor or a sense of resistance, and a dull tympanitic note on percussion.

The *symptoms of the acute exacerbations* resemble those of the primary attack. These acute exacerbations may occur at any time, so that the patient becomes afraid to travel from home, being apprehensive of the increasing severity of future attacks, or may become unable to pursue business or laborious occupation.

The **duration** of the disease varies from one to ten years; the acute attacks vary in number from two to twenty or thirty.

The **prognosis** depends upon the character of each acute attack.

The **treatment** is surgical. The question whether to operate during an interim or to wait until the symptoms of an acute attack call for surgical interference should be left to the surgeon to decide upon the merits of each individual case.

ULCERATION OF THE INTESTINE.

The following intestinal ulcerations may be described:

1. *Round ulcer of the duodenum* resembles gastric ulcer in its cause and appearance, but is much less common (as 40 to 1). The ulcer is usually single and situated near the pylorus. Localized peritonitis with adhesions, localized peritoneal abscess, perforative peritonitis, and hemorrhage are the most frequent complications, while stenosis of the pylorus, of the orifices of the common bile-duct, or of the pancreatic duct may result from cicatricial contraction.

The *symptoms* closely resemble those of gastric ulcer. Pain is referred to the right hypochondrium, is rarely severe, and appears later after eating than does the pain of gastric ulcer. There may be only irregular, ill-defined feelings referred to the hypochondrium, with localized tenderness. Dyspeptic symptoms and vomiting are exceedingly rare.

Hemorrhage occurs in one-third of the cases. The blood may be vomited or be passed in an altered condition by the bowel, or the patient may die before the blood has time to appear externally. Many cases run a latent or obscure course, and in these cases death from hemorrhage or from perforation may be the first indication of serious disease, although in the latter case severe continuous pain usually precedes the rupture by several days.

The *prognosis* is more serious than that of gastric ulcer.

The *treatment* is that of ulcer of the stomach.

2. *Duodenal ulceration* may occur after extensive burns of the skin. The duodenum is congested and ulcerated in patches of an irregular form, the lesions appearing in from seven to fourteen days after the injury. The exact pathogenesis of these cases is unknown. Hemorrhage and perforation are the chief symptoms, and the patient almost invariably dies.

3. *Embolic ulcers* may result from embolism or thrombosis of a branch of the mesenteric artery. The emboli may arise from endocardial vegetation or from atheroma of the aorta, while the occurrence of thrombus is favored by atheroma of the mesenteric artery itself. The mesenteric vessels being terminal arteries, embolism or thrombosis leads to hemorrhagic infarction of a section of the intestinal wall, which rapidly undergoes necrosis. The peritoneum over the affected area is inflamed and may be the seat of perforation, and the intestine itself in the vicinity is congested and infiltrated with blood. The diagnosis is to be made by attention to the following points: (1) The presence of a cause for embolism; (2) the presence of emboli in other organs; (3) symptoms of intense enteritis; (4) symptoms of peritonitis. If the embolus be septic, extensive suppuration of the intestinal wall will result. Embolic ulcers of the colon are exceedingly rare.

4. *Ulcers due to amyloid degeneration* may be found in any part of the intestinal tract, being due to local disturbances of nutrition consequent upon the diminished supply of blood that necessarily results from the waxy changes in the wall of the terminal arteries. These ulcers show no disposition to heal.

5. *Catarrhal and follicular ulcers* result from acute and chronic enteritis. Catarrhal ulcers are usually found in the colon, either as slight erosions or involving large areas by their extension and confluence. The floor and the walls of long-continued ulcers become greatly thickened. The natural termination is by cicatrization.

Follicular ulcers may be found in either the large or the small intestine. They are of round shape, with undermined edges. They may be so numerous that the bowel is studded by them. Follicular ulcers may extend by ulceration of neighboring parts or may perforate. Cicatrization seldom results if the lesion be extensive.

6. *Stercoral Ulcers.*—The pressure of hardened feces leads to necrosis and subsequent purulent infiltration of the mucosa. Stercoral ulcers occur in situations in which fecal accumulation is liable to occur, as in the cæcum, the rectum, the flexures of the colon, or above the point of stricture in intestinal obstruction.

7. The ulcers of typhoid fever, diphtheria, variola, and anthrax have elsewhere been described.

8. *Tubercular ulcers* are of common occurrence. They may appear as a primary infection from the ingestion of tubercular meat or milk, especially in children, but they are more common as the result of secondary infection complicating pulmonary or genito-urinary tuberculosis. The process begins first in the ileum, extending thence to the rest of the small and the large intestine. Tubercles first develop in the solitary or agminated follicles, undergo cheesy degeneration,

and break down to form ulcers. These follicular ulcers extend by suppuration and by extension of tubercular deposits along the line of the lymphatic vessels, so that "girdle" ulcers are formed, encircling the intestine at right angles to its long axis. The peritoneum covering the site of the ulcer is studded with tubercles, is coated with fibrin, and is adherent to adjacent surfaces. Perforation may occur; it is usually prevented, however, by the formation of peritoneal adhesions. The mesenteric glands are almost invariably enlarged and tubercular. The girdle shape of the ulcer distinguishes it from typhoid ulceration. A differential diagnosis in the earlier stages can be made by the fact that in typhoid fever ulceration of a Peyer's patch is uniform, whereas in tubercular disease separate follicles are involved, while others entirely escape. Cicatrization is rare, but it is possible.

9. *Leukæmic ulcers* result from necrosis of lymphoid deposits in the wall of the intestine. They are rare, however, except during the course of acute leukæmia.

10. *Scorbutic ulcers* may follow hemorrhages into the mucosa.

11. *Syphilitic ulcers* are rare in the small intestine except in new-born syphilitic children. Gummata of the intestinal wall may occur, and ulcers may result from their breaking down. Syphilitic ulceration of the rectum is not uncommon, especially in women; it leads to progressive fibrous stricture.

12. *Uræmic ulcers* may occur with advanced nephritis in several ways: (*a*) Ulceration of solitary and agminated follicles, with catarrhal entero-colitis; (*b*) as a result of a pseudo-membranous enteritis; (*c*) gangrenous ulceration may occur.

13. *Mercurial ulcers* follow pseudo-membranous enteritis from poisoning by mercury.

14. *Cancerous ulcerations* may result from the breaking down of submucous nodules.

15. Ulceration from external perforation may occur from ulceration and erosion of new growths or by the perforation of a neighboring abscess into the intestine.

The **symptoms** of ulceration of the intestine depend upon the position and extent of the ulceration and upon its pathological character.

Diarrhœa is a frequent symptom, being regularly present with ulcers of the lower portion of the colon and the upper portion of the rectum. Ulcers limited to the small intestine, the cæcum, and the ascending colon do not of themselves cause diarrhœa.

Hemorrhage varies in amount, the largest hemorrhages occurring with duodenal ulceration, typhoid fever, and perforation from without the intestine. If the origin of the hemorrhage be in the upper portion of the intestine, the blood is usually dark and altered and mixed with feces; hematin-crystals may alone be detected.

Pus in the stools is rare unless from ulceration, and hence its presence is of great diagnostic importance. Large evacuations of pus indicate rupture of a neighboring abscess into the intestine. Usually the quantity of pus is small, necessitating for its detection close scrutiny, and even microscopical examination, of the feces. Pus with blood and mucus usually indicates dysentery or an ulcerating carcinoma of the colon or the rectum. Shreds of tissue, if proven not to consist of undigested food, afford conclusive proof of rapid and extensive ulceration. Tubercle bacilli in the stools are usually, but not invariably, found in cases of tubercular ulceration.

Pain is frequently absent. It may be of the nature of a colic, or there may be steady pain due to a complicating localized peritonitis. Tenderness, which may be constant over a small area, is of value in localizing the seat of ulceration. Tenesmus occurs only if the rectum be ulcerated.

Fever depends upon associated conditions. Emaciation depends upon the extent and pathological character of the ulcers, and is more pronounced when the small intestine is affected.

Not infrequently ulcers of the intestine run an entirely latent course, and are unexpectedly found at post-mortem examination.

The complications of ulceration comprise localized peritonitis, peritoneal abscess, purulent or perforative peritonitis, and hemorrhage; should cicatrization occur, intestinal obstruction may result.

Treatment.—The diet should be easily digestible, nutritious, and unirritating. A milk diet is indicated in severe cases, and prolonged rest in bed may be necessary to accomplish good results. For ulceration of the small intestines antiseptics by the mouth should be given, to keep the intestinal tract disinfected so far as possible. Bismuth salicylate and subnitrate (each 15 grains every three or four hours) are of great value, but salol, bismuth subgallate, naphthalin, and resorcin may be used. For ulceration of the colon large injections of warm salt-solution are to be given to cleanse the bowels. The addition of astringent or irritating drugs, such as nitrate of silver, salicylic acid, or thymol, does not seem to increase the medicinal value of these injections, and certainly renders them painful and annoying.

Ulceration of the rectum can be treated, if within reach, by the methods pursued in treating external ulcers.

CANCER OF THE INTESTINE.

Carcinoma of the intestine usually occurs as a primary growth, and comprises from 4 to 8 per cent. of all cases of cancerous disease. Four varieties are encountered:

scirrhus, encephaloid, colloid, and the cylindrical-celled epithelioma. Growths in the large intestine are nine times as common as those of the small intestine, the seats of selection being the rectum (80 per cent. of all intestinal cancers), the sigmoid flexure, and the caput coli. Next in frequency comes cancer of the duodenum.

Pathology.—Scirrhus usually produces a hard infiltration of the intestinal wall, narrowing the lumen of the gut. The encephaloid and cylindrical-celled epithelioma form annular constrictions, large fungoid masses projecting into the cavity of the intestine, and are very prone to ulceration and hemorrhage. The colloid form produces a gelatinous infiltration of the intestinal wall, without much tendency to ulcerate or to cause obstruction. Secondary deposits are not uncommon, especially in the liver, the general rule being that when secondary cancer of the liver develops, the primary cancer in the intestine ceases to grow and often gives no further symptoms, so that the case will resemble one of primary cancer of the liver.

From the ulceration of the cancer perforation may occur into the peritoneum or into hollow viscera, forming fecal fistulæ; or extensive hemorrhage may result. The intestine becomes more or less occluded, the obstruction being often rendered more complete by fecal accumulation at the point of stricture.

CANCER OF THE RECTUM.

Symptoms.—Pain is usually more marked than that produced by cancer in any other part, excepting cancer of the tongue. The pain is not always of the same kind. (*a*) In some cases the pain is the same as that produced by cancer in any other part of the body—dull, boring, and continuous. (*b*) The pain may be neuralgic, and is due to pressure on the sacral plexus. These cases are frequently treated for sciatica.

(*c*) The pain may be due to obstruction of the rectum, being paroxysmal and straining in character. These cases are often treated for chronic dysentery or for fecal impaction.

Hemorrhage is usually in small amounts; it is brought on by straining attempts at stool. Occasionally, however, the bleeding is profuse.

In nearly all cases there is an irritating discharge which excoriates the anus and the neighboring parts. Should the sphincter ani be relaxed, as often happens, the condition of the patient is rendered more uncomfortable. There are regularly changes in the action of the bowels: (*a*) There may be diarrhœa accompanied by pain and tenesmus; (*b*) the stools may be deformed by being forced through the constricted rectum, so as to be ribbon-shaped or as small in diameter as a lead-pencil; (*c*) there may be constipation with symptoms of intestinal obstruction. Should the obstructing growth ulcerate, the constipation will suddenly give way. Cancerous cachexia intervenes, and the symptoms of secondary deposits in adjacent viscera or in the liver may complicate the latter stages of the disease.

Physical Examination.—There may be a hard ring felt two or three inches from the anus, usually just large enough to admit the finger (Fig. 48, A). In some cases the bowel is invaginated, so that the ring is pushed down by

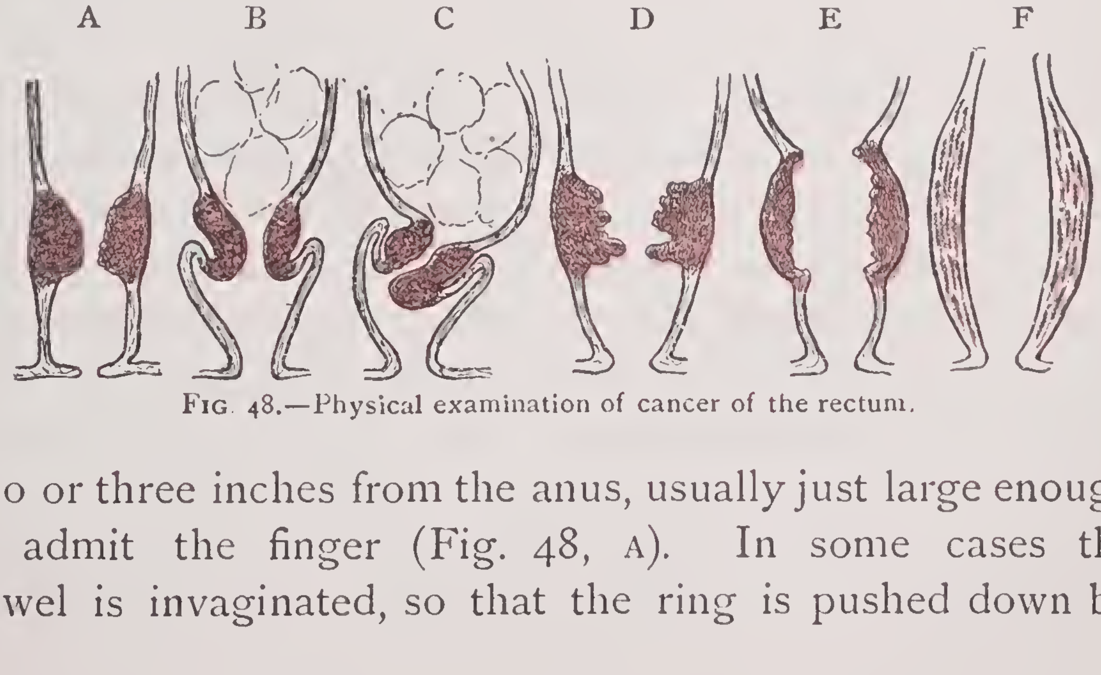

FIG. 48.—Physical examination of cancer of the rectum.

the accumulation of feces above (B), and the orifice of the constriction may be tilted so that it is found with difficulty (C). In either case there are felt projecting into the rectum large friable masses which bleed readily (D), or ulcerations with hard edges and floor may be detected (E). In cases of colloid carcinoma the wall of the rectum loses its soft, velvety feeling and becomes dense and thick, but no obstruction and no ulceration can be appreciated (F). In examination for rectal carcinoma the patient should stand up and bear down, to bring the growth within reach. If nothing can then be felt, examination under an anæsthetic is indicated.

Prognosis and Treatment.—An early diagnosis should be made if possible. Neglected cases run a fatal course of from two to four years, but life may be prolonged by an early excision of the neoplasm, as by Kraske's operation. In some cases a radical cure has thus resulted. Lumbar colotomy with the formation of an artificial anus may be indicated to relieve the constipation and to modify the pain.

CANCER OF THE CAPUT COLI.

The **symptoms** of cancer of the caput coli begin gradually and are at first obscure. There is a gradual loss of flesh and strength, with varied digestive disturbances. In other cases pain is the first symptom noticed. The pain may be dull and boring or sharp and colicky. If there be obstruction, pain is increased and symptoms of intestinal obstruction gradually appear. Physical examination reveals a *tumor* in the cæcal region—a tumor hard and irregular, either globular in shape or ovoid, its long axis agreeing with the course of the ascending colon. The tumor, which is usually adherent to the posterior abdominal wall, so that it is not apt to be movable, is most liable to be mistaken for fecal impaction or for a chronic appendicitis. The patient becomes cachectic, loses flesh and strength, suffers from diarrhœa or constipation, and dies exhausted or from intestinal obstruction.

Treatment of cancer of the caput coli is merely palliative in cases in which the diagnosis is made too late to allow of resection of the intestine.

CANCER OF THE DUODENUM.

Cancer of the duodenum usually presents itself as a movable tumor which cannot be differentiated from malignant growths of the pylorus. The tumor may be displaced into the lower abdominal region by reason of the weight. Dilatation of the stomach usually results, and the pancreatic duct and the bile-ducts may be obstructed.

The **treatment** is palliative.

NON-CANCEROUS TUMORS OF THE INTESTINE.

Non-cancerous growths are so rare that they possess only pathological interest.

Mucous polypi may occur, especially in children and in the rectum; or there may be found pediculated fibromata. Lipoma, sarcoma, lymphangioma, and myoma have been described.

INTESTINAL OBSTRUCTION.

Etiology and Pathology.—1. *Internal strangulation*, or "internal hernia," is the cause in one-third of the cases, and is the most frequent cause of obstruction in adults; 70 per cent. of the cases occur in males. The strangulation may be produced in various ways: A loop of intestine may be constricted by passing between peritoneal adhesions or by passing through apertures in the mesentery or the omentum, through the foramen of Winslow, or even through the diaphragm. Should the tip of Meckel's diverticulum be adherent to the mesentery or to the abdominal wall, a ring will be formed, through which a coil of intestine may pass. In 90 per cent. of all cases the ileum is the portion of the intestine involved. Strangulation results in obstruction, ulceration, sloughing, and eventually in perforation.

2. *Intussusception*, or invagination, which occurs in one-third the cases, is the most frequent cause of obstruction in children, one-third of the cases occurring during the first year, and one-half of the cases before the tenth year. This condition arises whenever one portion of the intestine slips into an adjoining part as a tuck may be taken in the finger of a glove; it appears to be due to irregular peristalsis, the receiving layer being drawn up by contraction of the longitudinal fibres. In each intussusception three layers of intestine are brought in apposition: an outermost or receiving layer (the intussuscipiens), the middle or returning layer, and the inner or entering layer. The outer and middle layers are in contact by their mucous surfaces, the middle and the inner by their serous surfaces. There is thus formed a longitudinal tumor varying in length from several inches to as many feet, and the invagination extends at the expense of the outer layer.

The intussusception is invariably downward. The affected parts become swollen, congested, and perhaps ecchymotic. In recent cases the peritoneum is merely congested and the intussusception may readily be reduced, but in cases living for two or three days the peritoneal surfaces become so agglutinated by fibrinous adhesions that reduction is no longer possible. The invaginated portion may slough and be passed by the rectum, so that spontaneous cure may result. Three-fourths of the cases of intussusception are of the ileo-cæcal variety, in which the valve slips into the large intestine, gradually inverting, appearing at the rectum in extreme cases. Intussusception of the ileo-cæcal variety or confined to the large intestine may last in rare instances for months without giving rise to acute symptoms.

3. *Volvulus*, or twist of the intestine, is the cause of the obstruction in one-seventh of the cases. Two-thirds of the cases of volvulus occur in men, and the condition is most frequent between the ages of thirty and forty. The twist, which is usually in the long axis of the intestine, is favored by a long mesentery, but one loop may be twisted around another or may be bent sharply upon itself. In one-half the cases the volvulus occurs in the sigmoid flexure, next frequently in the cæcal region. The twisting interferes with the circulation of blood and leads to necrosis of the affected portion of the intestine.

4. Acute obstruction from *strictures* and *tumors* may occur, but the obstruction is more apt to be chronic and progressive, although never complete. Narrowing of the lumen of the intestine may be due to the following conditions: (*a*) Congenital stricture as the result of fatal peritonitis, in which case the obstruction is usually in the rectum or the lower ileum; or there may be congenital malformation resulting in imperforate anus or rectum. (*b*) Cicatricial stenosis may result from previous ulceration. (*c*) New growths of the intestine, especially epithelioma. (*d*) Compression of the intestine by abdominal or pelvic tumors. (*e*) Contraction of inflammatory exudate, especially with tubercular peritonitis.

5. Obstruction may be due to foreign bodies within the intestine. Gall-stones may enter the intestine through the duct, or there may have been a communication between the intestine and the gall-bladder. Enteroliths may be formed by the phosphates of lime and magnesia being deposited about a central nucleus; or the foreign body may be a tangled mass of worms. Fecal accumulation is a common cause of obstruction, and will elsewhere be described.

Symptoms.—The three cardinal symptoms are pain, vomiting, and constipation. The pain may begin suddenly, while the patient is in apparently good health, and is usually localized, intermittent, and colicky. It soon becomes more intense and continuous, and spreads over the abdomen, being, however, more severe in its original situation. Tenesmus occurs only if the rectum be involved. Vomiting is constant and distressing. The vomited matters are at first gastric, then bilious, and finally stercoraceous. The higher up the obstruction the earlier does the fecal vomiting occur; but true fecal vomiting cannot occur with obstructions above the upper third of the ileum. The vomiting may cease at the approach of the fatal issue, or it may be replaced by distressing and obstinate hiccough. Constipation is usually preceded by the emptying of the intestine below the seat of obstruction. There is also an absence of flatus. In intussusception there may be discharges of mucus and blood. If the obstruction be low down in the intestinal tract, the abdomen is greatly distended and active peristalsis is visible, but if the obstruction be higher up these symptoms are not present. At first the abdomen is insensitive; later, exquisite tenderness is developed. The face becomes pallid and anxious; there is incessant thirst; the pulse is feeble and either rapid or slow; the urine is diminished or suppressed. There is not apt to be fever; on the contrary, the temperature may be subnormal. Symptoms of collapse or of peritonitis appear, and the patient rarely lives beyond the fourth or the sixth day.

Diagnosis.—The determination of the *seat of the lesion* is often extremely difficult. If the obstruction be in the duodenum or the jejunum, vomiting occurs early, collapse is rapid, there is no tympanites, and the urine is usually suppressed. If the lower ileum or cæcum be obstructed, the abdomen is distended in the umbilical region, the flanks are flattened, and active peristalsis can be seen. If the colon or the rectum be occluded, the flanks become likewise distended, there may be tenesmus, and the symptoms are not as severe as in the preceding forms. If four quarts of water can be injected slowly so as to fill the colon and the cæcum, the obstruction must be in the small intestine.

Diagnosis of the Course of the Obstruction.—Intussusception is essentially a disease of children. A sausage-shaped tumor usually occurs before the third day in the region of the ascending or transverse colon. There is usually tenesmus, with the passage of bloody mucus. Examination by rectum may reveal the lesion. Fecal vomiting is not common, and abdominal distention occurs in only one-third of the cases.

Strangulation is not common except during adult life. There is usually a history pointing to the presence of peritoneal adhesions. Pain is excessive; vomiting is incessant and soon becomes fecal; prostration rapidly becomes extreme. There is absolute constipation, but no tenesmus, and there is no tumor to be detected.

Volvulus is diagnosed with great difficulty, although it may be suspected if the sigmoid flexure be involved. As twists of the sigmoid flexure are often produced by the weight of accumulated feces, a history of fecal accumulation often precedes the accident.

Foreign bodies usually lodge at the ileo-cæcal valve. There may be the history of some bulky object swallowed, or of the passage of previous gall-stones.

For the diagnosis of *fecal impaction* see pp. 534, 535.

The diagnosis should also be made from functional obstruction occurring in hysterical patients, and usually following blows upon the abdomen, peritonitis, or the reduction of a hernia. The lesion seems to be a temporary cessation of peristaltic movements, as the result of which the downward advance of the intestinal contents is checked. In every case external strangulation by hernia must be excluded by careful search. Acute hemorrhagic pancreatitis may so closely resemble intestinal obstruction that a differential diagnosis cannot always be made.

The **prognosis** of every case of obstruction is grave, and usually is absolutely unfavorable unless the obstruction be relieved by surgical measures. Spontaneous cure of intussusception has been mentioned, but few children live long enough for the result to take place. Spontaneous cure may result in exceptional cases of strangulation by the formation of a fistula between two coils of intestine.

Treatment should be prompt and energetic. Purgatives are absolutely contraindicated. The patient should be put under the influence of opium almost to the point of semi-narcotization, the respirations being kept between 10 and 14 by its repeated administration. By this treatment all peristaltic action is checked, so that the gut will have a chance to untwist or to free itself from obstructing bands or from its invagination. The vomiting and pain are also checked, and the danger of collapse is lessened.

Washing out of the stomach with warm water may be repeated three or four times a day. In some cases this treatment has been useful in diminishing peristalsis and in lessening the abdominal pressure above the obstruction. The colon should be flooded with warm water, the patient being in the knee-chest position and preferably under the influence of an anæsthetic. Inflation of air may be practised, the air being introduced by a Davidson syringe, or the rectal tube may be attached to a siphon of carbonated water. These latter methods are most useful in cases of intussusception, but they are not devoid of danger, as rupture of the bowel has resulted when undue force has been used. Inflation and injection of water are of no service should the intussusception last longer than forty-eight hours, as by that time inflammatory fixation will have occurred.

These medicinal measures should not be continued after forty-eight hours, nor in any case in which the symptoms are rapidly becoming urgent, but laparotomy should be performed at once. Laparotomy should be done as early as possible in cases of strangulation, volvulus, and impaction of foreign bodies, before the interference with the circulation has led to sloughing of the intestinal wall.

HEMORRHAGE FROM THE INTESTINE; ENTERORRHAGIA.

Etiology.—Hemorrhage from the stomach is a symptom that may be produced in a variety of ways:

1. By ulceration of the intestines of any form.

2. By irritant and corrosive drugs.

3. By local injuries, such as those inflicted by foreign bodies, by hardened fecal masses, and by intestinal parasites, especially by the anchylostoma duodenale and the distomum hæmatobium.

4. By active congestion in severe inflammations, in intussusception and volvulus, and following the reposition of an incarcerated hernia.

5. By passive congestion with disease of the heart and lungs, with obstruction in the portal circulation and with hemorrhoids and venous varices.

6. By diseases of the blood-vessels, especially amyloid degeneration, or by aneurysm or embolism of a branch of the mesenteric artery.

7. By tumors of the intestine, especially cancer and polypi.

8. By causes without the intestine, as when blood enters the intestine from the stomach or from a ruptured aneurysm of the abdominal aorta.

9. By constitutional diseases, especially pernicious anæ-

mia, leukæmia, pseudo-leukæmia, scurvy, purpura hæmorrhagica, septicæmia, profound jaundice, yellow fever, acute yellow atrophy of the liver, and poisoning by phosphorus. Hemorrhage may also occur in conditions of hunger and inanition.

10. Hemorrhages in the new-born (melæna neonatorum) may be due to acute fatty degeneration or to syphilitic degeneration of the blood-vessels, to hæmophilia, or to puerperal infection.

Symptoms.—The blood may be vomited or be passed by the bowel, or the patient may die before the blood appears (concealed hemorrhage). Blood from the rectum or the sigmoid flexure is bright red, and its passage is accompanied by straining. Blood from the lower bowel is also smeared over the fecal masses. Blood from the ileum is usually dark red, the normal color generally being restored by adding water to the stools, and its passage is accompanied by increased peristalsis and diarrhœa. Blood from the jejunum and the duodenum is dark and tarry from the change of the hæmoglobin into hæmatin. These dark stools may resemble those produced by eating huckleberries or by taking iron or bismuth, but the differential diagnosis is to be made positively by the spectroscope and by the finding of hæmatin-crystals in the stools.

The general symptoms are those of hemorrhage in general. Following the hemorrhage the bowels may be obstinately constipated and there may be a high temperature (septic fever or resorption-fever).

Treatment.—Absolute bodily and mental rest must be enforced, and no food should be allowed for from twenty-four to forty-eight hours. Opium should be given in doses sufficient to check peristalsis; this drug is also of service in controlling restlessness and collapse-symptoms. Ice applied to the abdomen, so often recommended, is not only useless but actually harmful, tending, as it does, to stir up peristaltic action. For large hemorrhages astringent drugs by the mouth do no good, but in repeated small hemorrhages ergotin, turpentine, erigeron, hydrastis canadensis, acetate of lead, gallic acid, and large doses of bismuth subnitrate are of great service. If the bleeding come from the rectum or low down in the colon, astringent injections may be employed, but they should not be resorted to as a routine measure, because of their tendency to quicken peristalsis. Tamponage of the rectum causes retention of gas, straining efforts, and increased intestinal peristalsis, and it should not be employed except in cases of bleeding from the lower portion of the rectum.

FECAL ACCUMULATION.

Etiology.—Fecal accumulation may be primary, or secondary to stricture of the intestine. In the primary cases there is usually the history of previous constipation, although at the time of examination the bowels may be loose. In other cases the accumulation results from weakness of expulsive efforts, and is common after prolonged illnesses, as typhoid fever.

Pathology.—The situation of the impaction is usually in the cæcum or in the rectum, but it may be at any part of the large intestine. The fecal masses may totally occlude the lumen of the gut, or they may be packed in the lateral pouches of the colon, leaving a passage through which normal stools may pass. If the accumulation be large, a tumor will be formed, which may give rise to pressure-symptoms, especially if the rectum be the seat of the impaction. The longer the fecal masses are retained, the harder they become, so that they may even resist the edge of a knife. The accumulation varies in amount, in extreme cases exceeding fifteen or twenty pounds in weight.

Symptoms.—Two distinct types of fecal accumulation are observed—a complete and an incomplete form.

COMPLETE FECAL IMPACTION.—(*d*) If the *cæcum* be the seat of impaction, the patient will suffer from the effects of constipation and will complain of pain and tenderness in the cæcal region. To these cases the term "stercoral typhlitis" is often applied. Examination will reveal the presence of a tumor—not soft, baggy, painless, and sausage-shaped, as ordinarily described, but hard, irregular, and more or less tender, so closely resembling the characteristics of a morbid growth that a diagnosis is impossible by examination alone. At some particular time the obstruction becomes complete. Constipation is absolute, not even gas being passed; the temperature rises to from 101° to 104° F.; the pulse is rapid and feeble. The abdomen becomes tender and tympanitic, and there is pain, either paroxysmal and colicky or like the exquisite pain of peritonitis. Respirations are rapid and thoracic. The case closely resembles one of appendicitis with general peritonitis, but in fecal impaction examination reveals a greatly increased peristalsis of the intestine, whereas in peritonitis all peristaltic action is checked. Unless relieved, gastric, bilious, and stercoraceous vomiting occurs; the patient is more and more prostrated, and dies with all the symptoms of intestinal obstruction.

The *prognosis* of this form of impaction is good if the case be properly treated. Relapses, however, are likely to occur.

Treatment.—In this form of impaction purgatives are absolutely contraindicated, as by the violent expulsive efforts of the bowel produced by their action the hardened mass is jammed more tightly into the distended intestine. Exactly the opposite treatment is indicated: opium is to be given in doses sufficient to check peristalsis and to relax the intestinal spasm at the seat of the impaction. Under this treatment the tumor can be felt to move along the colon from day to day. When the mass reaches the transverse colon, copious salt-water irrigations will bring away large quantities of hardened feces. When the impaction is once broken up opium should be discontinued and the use of laxatives should be begun, the best of these being castor oil in small repeated doses. Faradism and massage applied along the course of the colon are often of service in promoting the passage of the fecal masses. To prevent reaccumulation, strychnine must be given for months to counteract the enfeeblement of the intestinal wall, and the bowels should be kept freely open by appropriate medication.

(*b*) If the *rectum* be the seat of the impaction, there will result ineffectual attempts at defecation, with straining and tenesmus, so that fecal impaction should be suspected in every person, especially the aged and those who are convalescing from a long, weakening illness, in whom the symptoms of dysentery appear. Pressure on the uterus may cause uterine symptoms. There may develop neuralgia from pressure on the sacral nerves, seminal emissions, or nocturnal enuresis.

The *diagnosis* is readily made by rectal examination, which reveals the presence of hard scybalæ in the rectum.

Treatment consist in breaking the impaction mechanically and in removing the scybalæ by the fingers, by the handle of a spoon, or by repeated enemata.

INCOMPLETE FECAL IMPACTION.—This form of impaction is usually seen in elderly persons with atony of the colon. The fecal masses are packed in the lateral pouches of the colon, leaving a passage channelled through the centre. The prominent symptom is diarrhœa, the loose stools arising from the irritation of the large intestine above the impaction. Some patients become poisoned by the accumulation, run down, and become so prostrated that the case may actually resemble typhoid fever. The condition is to be suspected in every case of chronic diarrhœa in old people. Examination may reveal scybalæ in the rectum, or there may be a sense of resistance with some dulness over the descending colon.

Treatment consists of purgation and colon-irrigation to bring away the fecal masses. Checking the diarrhœa with astringents regularly aggravates the condition.

AMYLOID DEGENERATION OF THE INTESTINE.

Etiology.—Amyloid degeneration of the intestine occurs as a secondary change in phthisis, in prolonged suppuration, especially of the bones, and in constitutional syphilis.

Pathology.—The lesion involves the large and the small intestine and is especially marked in the lower ileum. The amyloid degeneration begins first in the walls of the smaller arteries, and in advanced cases may involve the whole thickness of the intestinal wall. Ulceration of the mucous membrane is not uncommon.

Symptoms.—The principal symptom is a chronic diarrhœa without fever (unless from the primary disease), pain, or tenderness. Blood and pus, if appearing in the stools, are indicative of ulcerations. The diagnosis is aided by the presence of the causative disease and by the finding of amyloid changes in other organs, as the liver and the spleen.

4. DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Etiology.—Peritonitis may be primary or secondary.

1. *Primary* or *idiopathic peritonitis* is exceedingly rare. It may develop after exposure to wet and cold, or as a terminal event in Bright's disease.

2. *Secondary peritonitis* follows infection from inflammation or perforation of any of the organs covered with peritoneum. It thus may follow—(*a*) Penetrating wounds and laparotomies. (*b*) Rupture or perforation of any of the abdominal viscera. (*c*) Rupture of an abdominal abscess, such as appendicitis or suppurating inflammation of the Fallopian tubes. It has also followed rupture of an apparently normal Graafian follicle or extra-uterine gestation. It has also occurred after perforation of the diaphragm in empyema. (*d*) Extension from inflammation or ulceration of the stomach or intestines, cancer or suppurative inflammations of the spleen, liver, pancreas, and retroperitoneal tissues, or strangulated hernia. (*e*) Pelvic conditions—septic uterine conditions, decomposing thrombi, etc. Infection may be carried, as in the case of gonorrhœa, through the Fallopian tubes without the tubes being involved.

Bacteriology.—The bacterium coli commune is one of the most frequent micro-organisms found in the peritoneal exudate, and is met with especially in peritonitis due to intestinal perforation. Next in frequency are the pyogenic micrococci, the streptococcus being usually associated with puerperal peritonitis, while the staphylococcus pyogenes aureus or albus is usually found in cases following laparotomy. The diplococcus pneumoniæ and the gonococcus have been found; the amœba coli may occur in the peritonitis accompanying amœbic dysentery.

Varieties.—There are three distinct varieties of peritonitis: 1. Acute peritoneal sepsis; 2. Acute diffuse peritonitis; 3. Acute circumscribed peritonitis.

Acute Peritoneal Sepsis.

In this form of sepsis, which is also termed "acute peritoneal septicæmia" and "perforative peritonitis," we have a simultaneous and rapid infection of the whole peritonea cavity after perforation of the stomach or the intestines, after rupture of large abscesses, or after septic penetrating wounds or laparotomies.

Pathology.—The peritoneum may appear normal, or may be injected and without its normal lustre. In the peritoneal cavity is a small quantity of sticky, non-purulent effusion, consisting chiefly of micro-organisms. This exudate may be found on the surfaces of the peritoneum, and cocci may be found in the lymph-spaces. Feces, contents of the stomach, or pus from a ruptured abscess may be present, according to the nature of the primary cause. If the patient live long enough, purulent inflammation follows and the lesions of acute diffuse peritonitis are found.

The **symptoms** are due to shock and to toxæmia from rapid absorption of ptomaïnes. There is usually a sudden sharp pain in cases of rupture or perforation, the pain ceasing as the patient passes into the condition of shock. The pulse becomes rapid and feeble; the breathing is rapid and shallow; the skin is cold and clammy. Slight cyanosis appears, and the patient is restless. The temperature at the onset falls, only to undergo a subsequent rise before death to 105° or 106° F. Death from toxæmia results in from twelve to fifteen hours in the majority of cases. In cases of milder infection the patient may survive long enough to develop the symptoms of diffuse peritonitis.

ACUTE DIFFUSE PERITONITIS.

This form of sepsis, which is also termed "acute general peritonitis," "purulent," "progressive," "progredient," or "exudative peritonitis," occurs when a general infection is not severe or sudden enough to cause death from toxæmia, or when successive areas of the peritoneum become in turn affected.

Pathology.—The intestines are inflated with gas and protrude through the post-mortem incision. The peritoneum is congested or pale and soggy in appearance, and is covered with fibrin or with fibrin and pus which render opposing peritoneal surfaces adherent. There is an exudation of serum in cases of mild infection, or of pus if the infection be more severe, the pus being thin and yellowish or thick and creamy or putrid. The amount of the exudate varies from half a liter to twenty or thirty liters. There may be admixed contents of stomach or of intestines in cases of perforation. Blood is not found except after penetrating wounds or laparotomies.

If the patient recover, the serum is absorbed, the fibrin and pus undergo emulsification and absorption, and the exudate becomes organized, so that the peritoneum becomes thickened and adherent. These connective-tissue adhesions interfere with peristalsis and may lead to internal strangulation.

Symptoms.—*Tympanites* is usually marked; it is due to the paralytic condition of the intestine. The inflation of the intestines may be so marked that the thoracic viscera are displaced upward, interfering with the breathing and the action of the heart. The abdomen in these cases is usually protuberant, but in some cases it is of natural size, although the abdominal wall is tense and hard. In other abdominal diseases, such as intestinal obstruction, tympanites is also present, but is associated with active peristalsis, whereas in peritonitis the association of tympanites with absence of peristalsis is distinctive. Over the distended abdomen a tympanitic note is obtained by percussion. Tympany over the normal liver-area is suggestive of gas within the peritoneal cavity.

Pain and tenderness are usually at first limited to the locality first inflamed, but later they become more general. The pain is very severe, with acute exacerbations. The patient lies motionless on the back, with the knees drawn up to relax the abdominal wall, and the breathing is rapid and thoracic. Restless movements of the arms are often in sharp contrast to the immobility of the body and the lower extremities. In progressive peritonitis fresh encapsulations of pus are marked by an extension of the pain, and over these freshly involved areas the note is dull on light percussion. It is important to examine the patient frequently and to remember the extent and location of these areas of dulness. In rare cases it is possible for peritonitis to exist without either pain or tenderness.

Vomiting is a frequent symptom. In some cases the vomiting appears to be due to irritability of the stomach or the diaphragm, and is accompanied by the muscular efforts of vomiting. The vomited matters are composed of food and bile-stained mucus. In other cases there is a regurgitation of gas from the intestine into the stomach, so that, without muscular effort, the gas is raised with a brownish or bilious fluid. In other cases, as death approaches there occurs, without effort, a regurgitation of a brown fluid which may possess a fecal odor. This sign is of serious import.

Constipation is the rule, and results from diminished peristalsis. In some cases, however, diarrhœa may exist from the transudation of serum into the cavity of the intestine. This diarrhœa is more common with circumscribed peritonitis, especially if it has lasted for some time.

The *temperature* is usually raised and runs an irregular course bearing no direct relation to the severity of the disease. As a rule, a high temperature indicates an extensive peritonitis, but a low temperature does not necessarily indicate a mild attack. The fever may rise to 102° or 104° F., but in some cases there occurs a sudden fall in the temperature with the appearance of collapse-symptoms, indicating the intervention of an acute peritoneal sepsis. Death soon occurs in these cases, and the temperature may be high again at the time of the fatal issue. A steady rise in temperature usually indicates a spreading peritonitis. In some cases with encapsulated collections of pus the temperature may become markedly remittent. Absence of fever is noted as a rare exception, especially in peritonitis of such acuteness and intensity that the symptoms merge into those of acute peritoneal sepsis.

The *pulse* is rapid and "wiry," being more rapid than can be accounted for by the fever. As a rule, the pulse gives reliable information as to the general condition of the patient.

The *appearance* of the patient is characteristic. The face is drawn and pinched; the nose is sharp and cold. The tongue has a tendency to become brown and dry even if the fever be moderate.

The *intellect* remains surprisingly clear even to the last, but there may appear periods of muttering delirium associated with the symptoms of the "typhoid state."

The **duration** of the disease is usually between two and seven days.

The **prognosis** is exceedingly grave. Spreading infection of the peritoneum may be recovered from after laparotomy and drainage; recovery without operation may follow cases of mild infection in which the effusion is chiefly fibrinoserous without much pus; but in all cases of diffuse peritonitis, however mild the inflammation may appear, a most guarded prognosis must be given. Cases of streptococcus infection usually die.

Diagnosis.—The following conditions are most apt to be mistaken for acute peritonitis:

1. *Hysterical peritonitis.* Here every symptom of peritonitis may be reproduced exactly, even the collapse, the

tympanites, and the fever, but other hysterical manifestations are usually present, the duration of the attack is longer, and there may be recurrences.

2. *Intestinal obstruction.* Here the cause is usually present (fecal accumulation, intussusception, or malignant growth), the temperature is not usually elevated, the vomiting is often stercoraceous, and intestinal peristalsis is increased.

3. *Acute hemorrhagic pancreatitis* may exactly simulate peritonitis, so that a diagnosis between the two conditions cannot be made.

4. *Ruptured tubal pregnancy* gives a previous history of cramp-like pains and cessation of menstruation.

5. *Rupture of an abdominal aneurysm* usually gives rise to rapid collapse and intense anæmic symptoms.

ACUTE CIRCUMSCRIBED PERITONITIS.

Etiology and Pathology.—Acute circumscribed peritonitis, which occurs in cases in which adhesions are sufficiently resistant to limit the infection, is more apt to occur with infection in the lower abdominal zone. The most frequent cases occur from inflammation of the appendix or from puerperal or gonorrhœal infection of the uterus and the Fallopian tubes. The rupture of an ulcer of the stomach may lead to a circumscribed peritonitis within the lesser peritoneal cavity. There may thus form beneath the diaphragm a large air-containing abscess to which the name "subphrenic pyo-pneumothorax" has been applied. If the localized abscess be small, the pus may eventually be absorbed, encapsulated, or calcified; extensive collections may burrow or perforate. Gradual infection of the peritoneal cavity results in progressive peritonitis, and acute peritoneal sepsis may result from the internal rupture of the abscess.

Symptoms are local and general. *Local symptoms* consist of pain, tenderness, and the presence of an inflammatory tumor. *General symptoms* at first are those of an inflammatory character—prostration and continuous fever. Later appear the septic symptoms of pus-absorption, irregular fever, chills, cold sweatings, diarrhœa, emaciation, and delirium at night. Ultimately septicæmia develops, with the symptoms of the typhoid condition.

The symptoms of the disease may at any time merge into those of acute peritoneal sepsis or of progressive peritonitis.

Treatment of Peritonitis.

Peritoneal Sepsis.—The treatment is that of surgical shock—by stimulation, external application of heat, and small doses of opium. If the patient's condition justifies the risk, laparotomy may be performed, perforations closed, and the peritoneal cavity cleansed with warm sterilized boric-acid solution.

Diffused Peritonitis.—Mild cases may be treated medicinally at the start, but it is advisable for a surgeon to be in constant consultation in the case, so as to be ready for surgical interference should the medical treatment be unsuccessful. The object of the medical treatment is to prevent intestinal peristalsis, so as to allow of the formation of adhesions to limit the infection. The drug *par excellence* is opium, and the amount in which it can be given is remarkable, as in peritonitis there exists a tolerance of the drug. Alonzo Clark's method was to give such doses as would keep the patient semi-narcotized, repeated doses being given as soon as the respirations exceeded twelve to the minute. In ordinary cases from 4 to 8 grains daily sufficed, but as much as 420 grains have been given in a single day. It is said, however, that the same tolerance does not exist for hypodermic doses of morphine as when the drug is given by the mouth. It is not now considered necessary to employ such heroic doses, but only as much morphine is given as will suffice to keep the patient free from pain. Larger doses than ½ grain every two or three hours are rarely required. Morphine even in these doses should always be given hypodermically.

Concentrated saline laxatives, however, may be given at the onset in cases following operations or septic conditions.

Local applications are often of great comfort and of unquestionable utility. For the earlier stages cold ice-bags or the cold Leiter coil is serviceable, but after five or six days hot applications seem to be preferable. The tympanites may be relieved by turpentine stupes or by the passage of a rectal tube. Lavage of the stomach may relieve the distention of the upper portion of the abdomen. Fitz recommends the frequent puncture of the distended bowel with a small hollow needle in extreme cases of meteorism, stating that the danger of extravasation or of the escape of gas into the peritoneal cavity is comparatively slight.

The use of saline purgatives has been recommended by Lawson Tait and decried by others. Certainly it would seem that the increased peristalsis would rupture fine limited adhesions, and the general use of laxatives is to be deplored. No harm results from constipation in peritoneal cases. Rectal injections may, however, be given to relieve the large intestine. In all cases of spreading peritonitis with urgent symptoms, surgical treatment is the only one that affords the patient any hope. There is more danger in waiting too long for operative interference than there is in operating too early in the disease, when surgical treatment may not be necessary.

Acute circumscribed peritonitis calls for surgical treatment to open and drain the abscess. The operation may be deferred in many cases until the limiting adhesions have had time to become firm.

CHRONIC PERITONITIS.

Etiology.—Chronic non-tubercular peritonitis may succeed an acute attack or may be chronic from the first. The most frequent cause appears to be repeated tappings for the removal of ascitic fluid; but the condition may occur with chronic diffuse nephritis or with long-continued abdominal or pelvic abscesses. The disease is more common in alcoholic patients. In some cases no definite cause can be assigned, but many of the so-called "idiopathic" cases ultimately are proven to be tubercular.

Pathology.—The peritoneum is thickened by connective tissue, and opposing surfaces are matted and massed together by firm, thick adhesions. In extreme cases the peritoneum is between one-fourth and one-half inch in thickness. In places there are congested patches covered with recent deposits of fibrin. The omentum, which is usually much thickened, is rolled up to form a sausage-shaped tumor lying transversely across the abdomen. The capsule of the liver or of the spleen may be thickened, contracted, and the volume of these organs correspondingly reduced. The mesentery is thickened and contracted. There may be but little serum, so that the process is described as "adhesive" or "proliferative peritonitis," but in other cases ("ascitic peritonitis") there is a quantity of liquid exudation, either free or encapsulated by adhesions. The ascitic form seems to be especially common in children. Chronic peritonitis may be diffused, or the process may be limited to a circumscribed area.

Symptoms are general and local.

General Symptoms.—There is a progressive loss of flesh and of strength by which the patient becomes finally reduced to semi-invalidism. The temperature may at times be slightly elevated.

Local Symptoms.—Pain in the abdomen is constant and annoying rather than actually severe. There is usually considerable tenderness on palpation. Disturbances in digestion are almost constant. The bowels are usually constipated, although periods of diarrhœa may occur from time to time. Distortions and flexions of the intestines may result in obstruction, or the common duct may be so twisted or compressed as to cause persistent jaundice. Acute exacerbations of the inflammation may occur, with moderate fever and a marked increase of the pain and tenderness.

In children between two and ten years of age a chronic peritonitis which cannot be traced to any cause is not uncommon. The ascites is considerable, but the symptoms are not extreme and recovery usually ensues.

The results of a **physical examination** are not always uniform, and depend upon the amount of the thickening and adhesions, the rolling up of the omentum, and the presence or absence of a serous exudate. If there be much thickening with matting together of the adhesions, the abdomen yields a doughy resistance to palpation, totally unlike the soft feeling of a normal abdomen. The whole abdomen may even appear to be filled with a resistant nodular tumor. If the omentum be rolled up, it may be felt as an irregular mass lying across the abdomen, and may be mistaken for the nodular edge of an enlarged liver. If there be free effusion, the abdomen will be distended, and dulness or flatness will be obtained over the dependent portions of the abdomen, as well as over the flanks in the dorsal decubitus, with a tympanitic note over the uppermost portions. By changing the position of the patient there is a relative change in the position of the percussion-notes. Fluctuation can readily be appreciated. Small encapsulated collections of fluid, surrounded by thickened and adherent intestines, may so closely resemble tumors that a differential diagnosis from cancerous peritonitis is not always possible.

The **duration** of the disease is months or years.

For the **diagnosis** from tubercular and cancerous peritonitis, see the articles treating of these diseases.

The **prognosis** is bad, the patients usually dying emaciated; but in some cases the disease may cease progressing, or recovery may follow operative treatment.

Treatment is properly surgical. Ascitic accumulations should be withdrawn by puncture of the abdominal wall. Laparotomy, with the breaking down of adhesions and the drainage of encapsulated serous effusions, is often of curative value. Medicinal treatment is symptomatic. It is claimed that benefit is derived from abdominal inunctions of mercurial ointment.

CHRONIC HEMORRHAGIC PERITONITIS.

This rare condition is analogous to chronic internal pachymeningitis. The peritoneum is thickened by connective tissue, and on its free surface are wide, thin-walled blood-vessels. By successive hemorrhages fibrin is deposited in layers, so that the thickening is increased. The process, which is usually circumscribed, is most frequent in the pelvic region.

TUBERCULAR INFLAMMATIONS OF THE PERITONEUM.

The following forms of tubercular inflammations of the peritoneum are described: 1. Acute tuberculosis of the peritoneum; 2. Acute tubercular peritonitis; 3. Chronic tubercular peritonitis.

Acute Tuberculosis of the Peritoneum.

In this form of inflammation the peritoneum is studded with tubercles as one of the lesions of acute miliary tuberculosis. The peritoneum is otherwise normal, and shows no coincident inflammatory change, although there may be an effusion of clear serum.

The clinical symptoms are latent or obscure. Pain and tenderness are rarely observed. The abdomen may be distended and may present the evidences of a peritoneal effusion.

ACUTE TUBERCULAR PERITONITIS.

Etiology.—This condition is seldom primary, but extension of the disease to the peritoneum usually takes place from the intestines, the lungs, the pleura, the mesenteric glands, the Fallopian tubes, or the genito-urinary tract in either sex.

Pathology.—There is a miliary tuberculosis accompanied by the ordinary products of inflammation. The peritoneum is studded with tubercular granules or plates. Elsewhere the membrane is congested and coated with fibrin or with fibrin and pus. There is usually an abundant effusion of either clear or turbid serum, occasionally hemorrhagic, rarely purulent.

The **symptoms** usually begin abruptly. There are more or less severe abdominal pain and tenderness, with the other local symptoms of a general peritonitis. The temperature runs an irregular course varying between 101° and 105° F., and is usually higher at night, although there has been observed an "inverse" temperature with evening remissions. The pulse becomes increasingly rapid and feeble. There is a progressive loss of flesh and of strength. There may be diarrhœa or constipation, or these conditions may alternate with each other. The "typhoid condition" ultimately develops.

Physical Examination.—The abdomen is distended and tympanitic. Fluctuation is detected in one-third of the cases, especially in the earlier stages of the disease. Irregular masses may be detected on palpation in protracted cases; these masses may be due (1) to a thickened and rolled-up omentum, (2) to encapsulated exudation, (3) to tubercular mesenteric glands, or (4) to retracted and thickened intestinal coils.

Diagnosis.—The symptoms are not equally prominent in all cases, but there is considerable variety in their relative preponderance. If the general outweigh the local symptoms, the case may closely resemble one of typhoid fever. The diagnosis may be rendered even more definite by a rose-colored eruption over the abdomen, resembling typhoid spots, which occasionally appears in tubercular peritonitis. If the local symptoms are the more prominent, the case may be regarded as one of non-tubercular peritonitis; but the diagnosis is to be made from the latter condition by the absence of a cause for non-tubercular infection, by the presence of tubercular disease elsewhere, especially in the organs enumerated under the heading of *Etiology*, and by the more protracted course of the disease.

The **duration** of the disease is from four to six weeks.

The **prognosis** is exceedingly bad, but is not absolutely hopeless.

Treatment should be surgical. The abdomen should be opened and drained. Operative treatment is more effectual in chronic cases, but in a few acute cases cure has resulted from such peritoneal drainage. Medical treatment is entirely symptomatic.

CHRONIC TUBERCULAR PERITONITIS.

Etiology.—The etiology of the chronic is the same as that of the acute form of tubercular peritonitis.

Pathology.—Two types are recognized:

1. *Tubercular Ascites.*—The peritoneum is thickened and is studded with fibrous or cheesy tubercles in granules or

in larger masses. The omentum is thickened and rolled up; the mesentery is retracted. There are but few adhesions. There is, however, an abundant serous effusion, giving rise to the symptoms and physical signs of ascites. The effusion is usually serous, but in rare instances it may be hemorrhagic or milky.

2. *Tubercular Peritonitis with Adhesions.*—All the viscera are matted together in one boggy mass, either by connective-tissue thickening and adhesions or by soft gelatinous fibrin. Fluid effusion is usually scanty and may be encapsulated. The retraction of the omentum and the mesentery is the same as in chronic non-tubercular peritonitis. Throughout the thickened peritoneum are old tubercles and cheesy masses. Fibroid transformation of tubercle is more common in the peritoneum than in any other locality of the body.

The **symptoms** of the tubercular resemble those of the non-tubercular form. There are, however, tubercular lesions elsewhere that modify the clinical picture of the disease, and the course of the tubercular form is more severe and uncompromising than that of the non-tubercular form. Some cases run a latent or an insidious course, and are accidentally discovered at a laparotomy or at the post-mortem table. In these cases the peritonitis is more apt to be circumscribed, and usually it is found in the pelvis, from infection through the Fallopian tubes.

Malignant disease of the peritoneum, as a rule, progresses more rapidly than tubercular peritonitis, and is secondary to malignant disease of some one of the abdominal organs that can readily be diagnosed.

The **prognosis** is grave, but is not so hopeless as might be expected. The brilliant results following laparotomy and drainage have made a favorable prognosis the rule.

Treatment.—It is claimed that from 70 to 80 per cent.

of the cases operated on have been cured, but evidence is wanting that even the majority of these cases were actually tubercular. In some cases merely an exploratory laparotomy in which the abdomen has been opened, inspected, and sewed up again at once has been productive of good results.

The medicinal treatment is essentially constitutional and symptomatic.

CANCER OF THE PERITONEUM.

Etiology.—Primary cancer of the peritoneum is exceedingly rare, although cases of primary colloid growths of large size have been described. Secondary growths occur in connection with malignant disease of any of the abdominal viscera. The disease, which is more common in women than in men, occurs during advanced life.

Pathology.—The peritoneum is studded with carcinomatous nodules of various sizes, the favorite seats for the deposits being the omentum and the mesentery, the peritoneum near the umbilicus, and Douglas's fossa. The nodules may be small and discrete or larger and confluent, so that tumors of considerable size may form. Unaccompanied by coincident peritonitis, the process is spoken of as "carcinosis." Usually, however, the peritoneum is the seat of a chronic inflammation; it is thickened, the omentum is rolled up, and the peritoneal cavity contains an effusion which may be serous, hemorrhagic, or milky from the fatty degeneration of the cancer-elements. In rare cases the effusion may become purulent. To this form of malignant deposits with associated inflammation the term "cancerous peritonitis" is given. The retroperitoneal and mesenteric glands are usually involved, and the inguinal glands may be enlarged. Perforation or fistulæ may result from the ulceration of the cancer-masses, and fatal hemorrhage may occur from ulceration within the bowel.

Colloid carcinoma involves the peritoneum diffusely, converting it to a thick, gelatinous mass, often of enormous size.

Symptoms.—1. There are symptoms due to the primary growth. Cachexia is usually evident before the peritoneum becomes involved.

2. Symptoms of chronic peritonitis are present. In many cases ascites and progressive emaciation are the principal complaints. Fever is usually present, but its course is similar to that of tubercular peritonitis. Pain and tenderness are usually more marked than with ordinary chronic peritonitis, but in rare instances the course of the disease is painless throughout.

3. Severe hemorrhages may result from ulcerations within the intestinal walls, or rapidly spreading peritonitis from perforation may develop. Cachexia, waxy pallor, and weakness increase with the progress of the disease.

Physical examination yields no distinctive signs by which a positive diagnosis may be made. If there be much effusion, the tumors may be so obscured that a diagnosis from chronic peritonitis is impossible. Usually, after tapping, multiple nodules may be felt, but these nodules may be mistaken for the encapsulated exudate of a chronic tubercular peritonitis. According to Osler, multiple nodules, if large, indicate cancer, particularly in persons above middle life, whereas nodular tubercular peritonitis is more common in children. The rolled-up omentum may be appreciated as an irregular mass lying across the upper abdominal zone; it has, however, no diagnostic value, as the same condition occurs in chronic tubercular or non-tubercular peritonitis. Examination by the rectum or the vagina should be resorted to in doubtful cases, as Douglas's fossa is frequently involved early in the course of the disease. Secondary nodules about the navel are highly suggestive of cancer.

The diagnosis will be confirmed if cancerous fragments be removed through the trocar at the time of tapping. A milky appearance of the fluid is suggestive, but is not absolutely diagnostic.

The further diagnosis from tubercular peritonitis has been spoken of under the latter disease.

If the growth be *colloid*, the results of physical examination are altogether different. There are no nodules and no ascites. The abdomen is symmetrically enlarged, often reaching enormous proportions. On palpation the abdomen is apparently filled with a semi-solid mass.

The **prognosis** is absolutely bad. As cancerous peritonitis usually complicates pre-existing visceral cancer, the duration of the disease is seldom more than a few months. Cases of primary growth run a longer course.

Treatment is entirely symptomatic. The fluid may be relieved by tapping if it produce discomfort, but the ascites rapidly returns. Opium should be given in doses sufficient to quiet pain.

ASCITES; HYDROPERITONEUM; ABDOMINAL DROPSY.

Etiology.—A serous exudate within the peritoneal cavity is common in all forms of chronic peritonitis, whether simple, tubercular, or cancerous. The term "ascites," however, should more properly be limited to a serous transudation from stasis, without inflammatory changes. The ascites may thus be due—(*a*) to obstruction of the portal vein, either in the terminal branches within the liver, as in cirrhosis of the liver, or in a larger trunk without the liver as from thrombus-formation, from external pressure by proliferative peritonitis, new growths, or abdominal aneurysms; (*b*) to general venous congestion arising in the course of chronic heart disease, emphysema, or interstitial pneumonia; (*c*) to hydræmic blood-conditions, as in Bright's disease or in advanced anæmia.

The ascitic fluid is pale yellow, with occasionally a greenish tinge, and is usually clear. The specific gravity varies between 1010 and 1015, although in cases due to cancer of the liver the gravity may be as high as 1023. The fluid contains albumin, has the chemical characteristics of blood-serum, and may form a delicate fibrinous clot on standing. Ascitic fluid may at times present a milky appearance. If this appearance be due to the admixture of chyle ("chylous ascites"), the fat is molecular; if due to layers of fat-globules, the name "adipose ascites" is often applied to the fluid.

Chylous ascites is due to injury to the thoracic duct by perforation, rupture, or by the filaria sanguinis hominis, or to its thrombosis or obliteration. In adipose ascites the fat originates from the fatty degeneration of cells, usually the product of a cancerous or tubercular peritonitis.

The **symptoms** of ascites are due to the mechanical weight and pressure of the transudation.

In ascites due to portal obstruction the fluid accumulates within the peritoneal cavity, sinking into the most dependent portions, while the abdominal organs that contain air float upon the fluid as far as their peritoneal attachment will allow. The fluid is freely movable, occupying the most dependent portions of the abdomen with every change of position. If the fluid does not thus move freely, but changes its position slowly and incompletely or remains immovable, inflammatory exudation is indicated. Pressure upon the iliac veins will cause secondary œdema of the lower extremities. In ascites due to diseases of the heart and the lungs or to hydræmic blood-conditions the abdominal effusion is but an added symptom of a general dropsical condition, and is associated with œdema of the lower extremities, and usually with hydrothorax.

Physical Signs.—In the dorsal position the abdomen is flattened in the umbilical region and bulges in the flanks. In the upright position the lower abdominal region is alone prominent. In extreme distention the whole belly is rounded and the skin of the abdominal wall is tense, shining, and may present pinkish striæ as in pregnancy. The navel is usually protuberant. Enlarged anastomosing veins are usually seen coursing over the abdominal wall.

Percussion gives dulness over the fluid and tympany over the overlying intestines. By changing the position of the patient the relative areas of these percussion-notes becomes correspondingly altered. Percussion is important in differentiating between the distention of ascites and that of extreme meteorism. In cases of moderate effusion dulness appears in the umbilical region when the patient assumes the knee-chest position.

Fluctuation is obtained by the transmission of a wave from one side of the abdomen to the other by one hand being placed over one flank while the other flank is lightly tapped. In this way even a small amount of fluid maybe detected with great accuracy. The sign, however, may fail when there is great effusion under high pressure. In fat subjects a fluctuation-wave may run across the lax, flabby abdominal wall, but this superficial wave may be checked by pressing the ulnar edge of the hand firmly upon the linea alba.

The **prognosis** of ascites is dependent upon its cause.

The **treatment** is that of the primary cause. Stimulants should be given to sustain a failing circulation. Anæmic conditions require appropriate treatment. The fluid may be diminished by the free use of diuretics and cathartics, should the patient's strength allow of such treatment.

Eventually tapping becomes necessary for the comfort of the patient, although the operation may have to be repeated frequently, as the accumulation tends to recur. Care should be taken before tapping to exclude a distended bladder. The skin having been scrupulously sterilized, the puncture should be made with a straight trocar, in the median line midway between the symphysis and the navel, provided that previous percussion has not revealed an intestinal coil lying directly under the site of puncture. When the fluid ceases to flow the trocar is to be withdrawn, the opening being closed by a suture over which is applied an antiseptic pad. A tight many-tailed bandage applied during the tapping will facilitate the flow, and after the trocar is withdrawn will yield support to the relaxed abdominal wall; this application should never be omitted. The dangers of tapping are syncope, perforation of the intestine, infection of the peritoneum with unclean instruments, and hemorrhage from puncture of an artery of the abdominal wall.

5. DISEASES OF THE LIVER.

FUNCTIONAL DISTURBANCES OF THE LIVER.

To appreciate the various symptoms caused by functional disturbances of the liver it is necessary to consider the normal functions of the liver, and to see what symptoms will necessarily arise from the perversion of each function.

1. From disturbed glycogenic function the bodily heat is lowered, so that the patient becomes susceptible to cold; glucose may pass the liver unchanged and may appear in the urine (glycosuria).

2. Destructive metamorphosis of albuminoid matter not being properly performed, uric acid and sub-oxidized urea compounds are retained in the body, giving rise to headache, vertigo, mental dulness, and despondency. Muscular

or articular pains are common. The kidneys may become irritated by the uric acid and the oxalate of lime, and a chronic nephritis may develop. In some cases renal calculi result.

3. Defects in the quantity or the quality of the bile allow of general malnutrition. Fats not being easily absorbed, the patient becomes thin. There are constipation and intestinal flatulence.

4. From the failure of the liver to destroy the poisons arising in the normal processes of digestion (peptotoxines), or the alkaloids of intestinal fermentation, these toxic products pass the liver unchanged, and a general toxæmia is the result.

Etiology.—Functional disturbance of the liver may be primary or secondary.

Primary cases arise—(1) From errors in diet. The food may be too rich, too excessive, or too abundant in fatty and saccharine ingredients, or there may be an over-indulgence in malt liquors. (2) From want of exercise and from deficient oxidation-processes.

Secondary cases arise—(1) From structural changes in the liver. (2) From disorders of gastric or intestinal digestion. (3) From disorders of the heart and the lungs, interfering with a proper circulation of blood within the liver and with oxidation-processes. (4) Some cases seem to be due to mild malarial poisoning.

Symptoms.—Individual symptoms may be inferred from the consideration of the preceding paragraphs. Clinically the cases may be divided into two groups:

1. The whole nutrition of the patient is below par; he looks anæmic and imperfectly nourished, and the complexion has a muddy, sallow tinge. The tongue is furred and flabby. The appetite is generally lost, especially during the earlier portions of the day. The bowels are con-

stipated, the stools being dryish and clay-colored. There is a disagreeable taste in the mouth, especially in the morning, variously described as "bitter" or "pappy." The breath is usually offensive. Headaches are frequent; they may be so persistent and severe as to suggest organic disease of the brain. Attacks of "sick headache" incapacitate the patient from time to time. The mental condition is one of apathy, with periods of irritability and depression. The urine generally shows deposits of urates, of oxalate of lime, or of uric acid. These patients are commonly described as "bilious."

2. Patients of the second group of cases do not become emaciated nor do they lose strength. The intestinal symptoms are slight, but the cerebral symptoms are prominent and distressing. Vertigo is often so pronounced that the patient is afraid to leave the house alone. There is loss of memory, with failure of the mind to concentrate itself for any length of time. Headache is frequent and distressing. There are alternate pallor and flushing of the face, with a sense of throbbing fulness in the head. The urine may contain oxalate of lime, uric acid or the urates, or may be normal. In these cases the symptoms are due to the vasomotor disturbances of the vessels of the brain, due to their irritation by toxic products of body-waste or of intestinal digestion.

The **prognosis** depends largely upon the will-power of the patient to carry out the necessary treatment.

Treatment.—Any dietetic error should be corrected. The food should be simple and wholesome, and fatty and starchy food should be reduced in quantity. Alcoholic stimulants should, in general, be interdicted. The most benefit is to be derived from active exercise in the open air to the point of moderate fatigue. The exercise, once begun, should be systematic, should be graded to suit the individual strength of the patient, and should be of such nature as to afford pleasure and enjoyment. The drug-treatment consists chiefly in controlling dyspeptic conditions, in giving laxatives when required, and in the use of cholagogues. Of the latter, podophyllin, rhubarb, ipecac, magnesium sulphate, hydrochloric acid, and salicylic acid are the most serviceable. The modified rhubarb and soda mixture, while unpleasant to the taste, is of great value:

℞.	Pulv. rhei,	gr. ij;
	Sodii bicarb.,	gr. v;
	Pulv. ipecac.,	gr. ¼;
	Tinct. nucis vomicæ,	♏v;
	Aq. menth. pip.,	ʒj.—M.

Sig. One teaspoonful three times daily, before meals.

CIRCULATORY DISTURBANCES OF THE LIVER.

Anæmia.—This condition, frequently found post-mortem, is not accompanied by any symptoms.

Active congestion was formerly regarded as a more important condition than at the present time. Physiological congestion occurs after a hearty meal. Acute congestion may occur with infectious diseases, especially malaria, typhoid fever, and dysentery.

The *symptoms* are not characteristic. There may be slight enlargement of the liver with tenderness on palpation.

Treatment is by active purgation.

Passive congestion (Chronic congestion; Nutmeg liver).—This condition occurs whenever there is a mechanical obstruction to the outflow of blood from the liver, during the course of heart disease with a failing right ventricle, with diseases of the lungs interfering with the flow of blood from the right to the left heart, and with pressure on the vena cava by thoracic tumors.

Pathology.—The central vein of each hepatic lobule is dilated, and the liver-cells in its neighborhood become pigmented and atrophied. The cells at the periphery of the acinus become fatty. The mottling of the reddish-brown depressed centre and the yellowish periphery of each acinus gives rise to the term "nutmeg" liver. In long-continued cases connective tissue may be deposited in and between the lobules, starting usually from the central vein. In the earlier stages the liver is enlarged, but the longer the congestion lasts, the smaller and denser the liver tends to become.

The *symptoms* are chiefly those of the primary lesion with the attending venous congestions. There may be added symptoms of a moderate degree of portal obstruction (see Cirrhosis of the Liver). Nausea, vomiting, and even vomiting of blood may occur, and slight jaundice may be evident in the conjunctivæ and in the urine.

The *treatment* is that of the original disease. Depletion of blood from the liver is accomplished by vegetable or saline laxatives. An occasional mercurial purgation by calomel or by blue pill is recommended.

DISEASES OF THE CAPSULE OF THE LIVER.

Acute Perihepatitis.

Etiology and Synonyms.—The cause of acute perihepatitis may be direct violence, but usually there is found perforation of an ulcer of the stomach or the duodenum, or infection or rupture of a neighboring abscess, especially of the liver, the gall-bladder, or the right kidney. *Synonyms:* Subphrenic abscess; Subphrenic pyo-pneumothorax.

Pathology.—The peritoneum of the liver and of the corresponding surface of the diaphragm is congested and covered with fibrin and pus. Adhesions form, allowing of a circumscribed peritoneal abscess (subphrenic abscess). The pus may be yellowish-red in color, from the presence in it of bilirubin, and it may contain crystallized fatty acids. If the cause be a perforation of an ulcer of the stomach or of the duodenum, the pus may be mixed with air (subphrenic pyo-pneumothorax).

The **symptoms** of acute perihepatitis are those of a localized peritonitis. There are pain and tenderness with the general symptoms of fever. In perforative cases the onset is abrupt and may be accompanied by shock. In general, the course of the disease is that of an empyema or an abscess of the liver. Drainage or evacuation of the pus may be followed by cicatricial contraction of the abscess-wall, so that compression may be produced upon the liver, the vena cava, the bile-duct, or the portal vein.

The **physical signs** resemble those of empyema or of an abscess of the liver. The right hypochondrium is distended and motionless. There may be a friction sound detected early in the disease. Later the friction disappears and is replaced by dulness or flatness and absence of voice and breathing.

Subphrenic pyo-pneumothorax may give physical signs similar to those arising from a like condition above the diaphragm.

The **diagnosis** of perihepatitis is readily made, should the aspirating-needle draw ochre-colored pus containing bilirubin and fatty acids. The etiology of the condition also gives a clue to the correct diagnosis.

Treatment is entirely surgical, consisting in opening and draining the abscess.

CHRONIC FIBRINOUS PERIHEPATITIS.

Etiology.—This condition may follow acute perihepatitis or may be chronic from the start, as the result of a chronic irritation of a neighboring inflammation or of long-continued pressure over the liver.

Pathology.—The peritoneum covering the liver is thickened and is adherent in places to opposed surfaces. Shrinkage of the fibrous capsule may result in atrophy of the liver or in constriction of the veins or the ducts.

Symptoms.—There are no characteristic symptoms. A friction rale may be heard over the liver during inspiration, but this sign is inconstant.

The **treatment** is that of the causative disease.

Syphilitic Perihepatitis.

The various syphilitic lesions of the liver will be considered under the heading of Syphilitic Diseases of the Liver.

ACUTE PARENCHYMATOUS HEPATITIS.

Synonyms.—Acute yellow atrophy; Malignant jaundice.

Etiology.—*Secondary* acute fatty degeneration of the liver may occur with many of the infectious diseases, in the latter stages of cirrhosis or of biliary retention, with yellow fever, and with phosphorus-poisoning.

Primary cases are exceedingly rare. Individuals under thirty years of age are more frequently attacked, and women, especially during pregnancy, are especially liable to the disease. At times several cases occur in one family or in barracks, showing an epidemic tendency. The actual cause of the disease is unknown, although it is supposed to be due to an unknown bacterial infection.

Pathology.—The liver is reduced from one-third to one-half in size, and its capsule is in folds and wrinkles. The organ is jaundiced or mottled yellow and red, the latter color representing the more advanced stages of the lesion, in which all fat has been absorbed from the liver-tissue. Microscopically are found extensive fatty degeneration and necrosis of the liver-cells, ending in their ultimate absorption. Crystals of leucin, tyrosin, and bilirubin may be found, and in most cases there is seen a small-celled infiltration of the stroma. The body-tissues are deeply jaundiced. The spleen is large and soft in about half the cases. The heart-muscle shows granular degeneration. There is acute degeneration of the kidney. There are numerous hemorrhages throughout the body.

Symptoms.—In the majority of cases there is a premonitory gastro-duodenitis, with nausea, vomiting, and jaundice. The onset of the disease itself comes suddenly and severely.

Nervous Symptoms.—There is violent headache. Active delirium, amounting to mania, may be so marked as to require physical restraint. The attacks of delirium become less and less severe and alternate with periods of stupor until finally the patient passes into the typhoid condition, with low, muttering delirium and extreme prostration.

Jaundice, if not developed during the preliminary period, now becomes noticeable. Its absence is rare. The sudden occurrence of jaundice with violent cerebral symptoms is suggestive of this disease.

Hemorrhages occur in the subcutaneous and submucous tissues, from mucous surfaces, and into internal viscera. Pregnant women abort with violent post-partum bleeding.

Urinary symptoms are somewhat characteristic. The urine is diminished, jaundiced, and contains albumin and casts. Urea is markedly diminished, and may be altogether absent in the latter stages, and in its place are found crystals of leucin and tyrosin. The former, occurring in the form of globules resembling fat, is seen only upon evaporation of the urine; the latter, in the form of needles, occurs as a spontaneous deposit. Leucin and tyrosin are not, however, constantly present in the urine of this disease.

General symptoms are not characteristic. The temperature may be normal, subnormal, or slightly elevated. A high ante-mortem temperature, however, is almost always observed. Vomiting is usually a prominent symptom, and blood may be raised. The bowels are constipated; the stools are clay-colored or may consist of blood. The pulse becomes rapid and feeble, the typhoid condition becomes more profound, and the patient dies, usually within a week, although the case may be somewhat more protracted.

Physical examination shows a rapid diminution in the size of the liver. As the flabby liver tends to drop back, there may finally be only a slight line of liver-dulness in the axillary region, and no dulness at all in front. If the disease attack a previously enlarged liver, the diminution will be less marked and the diagnosis will be more difficult. The liver is usually tender on palpation or percussion. The diagnosis is further aided by the presence of an enlarged spleen, by the jaundice, and by the hemorrhages.

The **diagnosis** may be made positively upon the association of severe jaundice, cerebral symptoms, diminished size of the liver, and the presence of leucin and tyrosin in the urine.

The case may be mistaken for one of phosphorus-poisoning, but in the latter condition there is the history of poisoning, the presence of phosphorus in the vomited matters, the onset is more sudden, gastro-intestinal symptoms are more marked, and leucin and tyrosin are absent from the urine.

The latter stages of hypertrophic cirrhosis may resemble acute yellow atrophy, but the liver is enlarged and the course of the disease is essentially chronic.

Conditions of profound jaundice with cerebral symptoms (cholæmia) may so closely resemble acute yellow atrophy that a differential diagnosis is very difficult.

Prognosis.—The disease is invariably fatal.

The **treatment** is symptomatic.

CIRRHOSIS OF THE LIVER.

Synonyms.—Chronic interstitial hepatitis; Fibrous cirrhosis.

Three forms of cirrhosis of the liver are recognized: 1. Atrophic cirrhosis; 2. Hypertrophic cirrhosis; 3. Syphilitic cirrhosis. The "Glissonian cirrhosis" has been described under the heading of Chronic Perihepatitis.

ATROPHIC CIRRHOSIS.

Etiology.—Fibrous disease of the liver is almost regularly due to the action of irritants brought to the liver by the blood-vessels. In over two-thirds of the cases the immediate cause is alcohol, especially the stronger liquors. There may be the history of excessive indulgence, or the habit of "nipping" may be confessed. In some cases indulgence in the lighter wines or in malt liquors may lead to the disease. Other irritants entering the liver by the portal vein may be adduced as causes of the disease—highly-spiced food and the ptomaïnes and other alkaloids of intestinal digestion—but upon this point satisfactory evidence is lacking. Cirrhosis may follow rickets, scarlet fever, or typhoid fever, or may result from long-continued passive hyperæmia of the liver. In coal-miners the disease may follow the swallowing of coal-dust, which is deposited as solid pigment in the liver. There has been described a senile form of cirrhosis analogous to other arterio-sclerotic changes in the different viscera.

Cirrhosis of the liver is far more common in men than in women, because of their more frequent indulgence in alcohol. It usually occurs between the ages of thirty-five and sixty years, and it is far less common in children than was formerly supposed, although in them the majority of cases are of syphilitic origin. It is a peculiar fact that cirrhosis is exceedingly rare in the negro, although in this race intemperate habits are common. Two autopsies only of this disease have been made at the Colored Hospital in New York during the past twenty-five years.

Pathology.—The liver is small, hard, and dense. Its weight may not exceed a pound or a pound and a half. The surface is granular, and the capsule is usually thickened. On section the organ may be bile-stained or may present grayish-white streaks of connective tissue. It was owing to the yellow appearance of the organ that the name "cirrhosis" (*κιῤῥός*, yellow) was first applied by Laennec. A primary increase of the size of the liver does not occur. Upon close examination the essential lesion is seen to consist of the formation of connective tissue within the liver, either loose and cellular or dense and fibrillated. The fibrous tissue usually extends about the radicles of the portal vein at the periphery of the acinus, giving the organ a finely granular appearance; or the connective tissue may surround groups of acini, resulting in a coarsely granular appearance. The tissue may extend into the liver from the capsule in large irregular streaks and bands, although this arrangement is more common in syphilitic cirrhosis.

Pressure-changes are seen in the liver-cells, the blood-vessels, and the gall-ducts. The liver-cells, especially at the periphery of the acini, undergo fatty degeneration and become atrophied or flattened by pressure.

The rootlets of the portal vein between the lobules are pressed upon and obliterated. This interference with the portal circulation gives rise to the important lesions of portal obstruction. There is usually an increase in the size of the hepatic artery furnishing blood to the new fibrous tissue. The gall-ducts may be the seat of a catarrhal inflammation or may become obliterated. In some cases there occurs an irregular production of new gall-ducts.

Secondary lesions result directly from the portal obstruction. The spleen becomes congested and hyperplastic. The stomach and the intestines are congested or show chronic catarrhal inflammation. Ascites results from the stasis, may reach an extreme degree, and may be accompanied by a mild form of chronic peritonitis. Hemorrhoids result from congestion of the veins of the rectum. There are evidences of collateral circulation between the branches of the portal veins and those of the vena cava, at the junction of the œsophagus and the stomach, along the sigmoid flexure and rectum, in the retroperitoneal plexus, and about the umbilicus. The radiating varicose veins about the latter situation have received the name of "caput Medusæ." The branches of the internal mammary and epigastric veins also become dilated and tortuous.

Associated lesions are not due directly to the cirrhosis, but are frequently found in the same patient, and arise from the same etiological factors as does the fibrous disease of the liver. These lesions comprise endocarditis, atheroma of the aorta, endarteritis, emphysema, and chronic diffuse nephritis. Acute tuberculosis is a not infrequent complication.

Symptoms.—There may be an antecedent alcoholic gastritis. When cirrhosis develops, the symptoms present themselves in four groups:

1. *Symptoms of functional disturbance of the liver.* The various intestinal, urinary, cerebral, and nutritive symptoms are present as in the cases previously described, and cirrhosis should be suspected if these symptoms occur in an alcoholic subject.

2. *Symptoms of obstructed portal circulation* are more distinctive. Gastro-intestinal symptoms are those of congestion or chronic inflammation, dyspepsia, vomiting, hæmatemesis which may be profuse or even fatal, constipation, and hæmorrhoids. Ascites gives rise to enlargement of the

abdomen and to dyspnœa; the transudation may press upon the iliac veins, causing œdema of the lower extremities. As long, however, as compensatory circulation is maintained, the obstructive symptoms are slight or absent.

3. *Symptoms of jaundice* are due to the obliteration or catarrhal inflammation of the bile-ducts or to gastroduodenal catarrh. The jaundice is rarely marked at first, amounting only to a muddy, sallow tinge. The facies is fairly characteristic: the skin is dry and of an icteroid hue; the nose and the cheeks show distended veins; the eyes are sunken and watery. From time to time attacks of more severe jaundice occur, and in some instances, especially toward the termination of the disease, the jaundice becomes extreme and is accompanied by cerebral symptoms.

4. *Symptoms of toxæmia* may develop at any time. The patient may become actively delirious, noisy, and talkative; or he may become stupefied, with periods of semi-coma and muttering delirium, or may even develop convulsions. These symptoms may be due to intense jaundice, to alcoholism, or to uræmia, while in other cases the exact nature of the toxæmia cannot be ascertained. The course of the disease is usually afebrile throughout, except that a moderate temperature may occur as a terminal symptom. At no time is there pain or tenderness over the liver.

Physical examination shows a diminution in the size of the liver, an enlargement of the spleen, and the presence of ascites and of anastomosing veins. A positive assurance that the liver is actually diminished in size is often lacking, as an apparent but not a real, diminution may be caused by the overlapping of the liver by an emphysematous lung, or by the liver being tilted upward by a distended stomach or colon; or the lower line of liver-flatness may be obscured by tympany from intestinal distention. In other cases the cirrhosis may occur in a liver previously enlarged by waxy or fatty change. The liver is thus rendered smaller than its previous size, but still may be larger than normal (see Hypertrophic Cirrhosis). The enlargement of the spleen is best determined by palpation, as the percussion-boundaries may be obscured by tympanites, by the solid or liquid contents of the stomach or the colon, or by the ascites. It is important to remember that the spleen may not be increased in size if there be continued diarrhœa or vomiting or if its capsule be thickened from perisplenitis.

Prognosis.—The course of the disease is protracted. The exact duration of any given case cannot be determined accurately, as the onset is insidious. If the patient abstains from his alcoholic habit, the lesion may not develop further, so that he may live for years. In ordinary cases the end is reached within a year or two after the diagnosis is made. Death may result from hæmatemesis, from Bright's disease, from delirium tremens, from the cerebral symptoms of severe jaundice, or from toxæmia and exhaustion. In the latter instance the patient becomes anæmic and emaciated, the enlarged ascitic abdomen being in marked contrast to the emaciated chest and extremities. Cerebral symptoms develop—stupor, muttering delirium, and semi-coma. The pulse becomes rapid and feeble, and death finally results.

Treatment.—The patient should abstain from alcoholic drinks and from highly-seasoned food. The diet should be of the simplest character, and the value of a prolonged milk diet cannot be overestimated. Exercise should be moderate and gradual; the skin is to be kept active by baths and frictions; the bowels should be kept open; and all errors of digestion should receive appropriate treatment. In patients with a syphilitic history mercury and potassium iodide may be given. Quinine and arsenic are indicated if the patient lives in a malarial locality. Cholagogues are indicated for the relief of the functional disturbances of the liver. When symptoms of portal obstruction appear, the indication for treatment is to deplete the engorged intestinal vessels by occasional catharsis. For this purpose from half an ounce to an ounce of magnesium sulphate may be given in a concentrated solution before breakfast, or any of the vegetable purges may be administered. An occasional dose of calomel or of blue pill is also of service. The ascites is to be relieved by diaphoretics, cathartics, and diuretics. Tapping should not be resorted to unless the ascites be distressing and be unrelieved by medicinal treatment, as the operation usually must be repeated frequently, and cerebral symptoms are apt to follow the withdrawal of the effusion. Repeated tappings, moreover, may result in chronic peritonitis. Toward the close of the disease alcohol may be necessary as a stimulant.

HYPERTROPHIC CIRRHOSIS.

Of hypertrophic cirrhosis three forms are described:

1. In the first variety the lesions are the same as in the atrophic form, but the increase of the connective tissue is greater than can be compensated for by the atrophy of the liver-parenchyma. The result is a cirrhotic liver of large size. The secondary lesions and the symptoms are identical with those of the atrophic form.

2. *Biliary cirrhosis*, or *hypertrophic cirrhosis with jaundice*, usually results from chronic bile-retention, and frequently follows chronic obstruction of the common duct by a gallstone, by cicatricial contraction, or by pressure from without. A primary form of biliary cirrhosis has been described. The bile-ducts become distended and are surrounded by new deposits of connective tissue. Fatty degeneration and atrophy of the liver-cells are not marked, the lesion differing greatly in this regard from that of the atrophic form.

The liver is symmetrically enlarged, extending to the umbilicus or even to the level of the anterior superior spines, is deeply jaundiced, and is resistant to the feeling and to the knife. Secondary lesions of portal obstruction are usually present, but to a less degree than in the atrophic form. Ascites is usually absent, although it may appear late in the disease. Jaundice is persistent and extreme throughout, yet the feces may be bile-stained. Hemorrhages in biliary cirrhosis are infrequent. At any time there may appear symptoms closely resembling those of acute yellow atrophy. The temperature rises to 102°, 104°, or even to 106° or 108° F., jaundice rapidly becomes more extreme, delirium and convulsions appear, and a fatal issue rapidly follows. Leucin and tyrosin, however, do not appear in the urine. In other cases death results from emaciation and debility.

The **course** of the disease is from three to seven years.

The **treatment** is that of cirrhosis in general. Good results have followed the frequent use of small doses of calomel, one grain being given several times a day for several days, and then discontinued should too free purgation follow or should stomatitis threaten, to be again administered after five or six days' interval.

3. *Fatty Cirrhosis.*—In this form of cirrhosis fatty and cirrhotic changes are found associated. The liver is large and resembles a fatty liver, but it is firmer, more resistant to the feeling and to the knife, and microscopically shows an increase in the connective tissue. The clinical course is that of atrophic cirrhosis, differing only in the fact that the liver is found to be enlarged. This form of cirrhosis is very common.

SYPHILITIC CIRRHOSIS.

Etiology.—Syphilitic disease of the liver may be the result of hereditary syphilis, occurring as a congenital manifestation or during childhood, or the liver may be affected as a tertiary lesion of the acquired disease.

Pathology.—Pathologically, three types of syphilitic disease of the liver may be described, but they are usually combined and blended with each other:

1. *Syphilitic Perihepatitis.*—The capsule of the liver is thickened and is adherent to opposing peritoneal surfaces. From the thickened capsule bands of connective tissue pass into the substance of the liver, forming fibrous scars that cause considerable deformity.

2. *Diffuse Syphilitic Hepatitis.*—The liver is large, firm, and resistant. Its color may be compared to that of sole-leather, or its appearance may resemble that of the amyloid liver. Microscopically the hepatitis does not present essential differences from the lesion of ordinary cirrhosis, although in some instances large groups of acini are surrounded by fibrous bands visible to the naked eye, and extensive puckered cicatrices may be found. Gummata are usually present at some stage of the disease. The lesions of portal obstruction are regularly present.

3. *Gummata* occur as nodules varying in size from that of a pea to that of a lemon. Fresh gummata are reddish-gray and of a translucent appearance. The older gummata show at their periphery a connective-tissue capsule; their centres may become cheesy, fibrous, or puriform, or they may be infiltrated with lime-salts. Smaller gummata undergo fibroid transformation and result in disfiguring cicatrices. Gummata alone cause neither portal obstruction nor jaundice, as a rule, unless they form in a locality where pressure on the portal vein or the bile-duct is possible.

Symptoms.—1. *Of the Perihepatitis.*—Pain and tenderness are commonly observed; the movements of the liver during respiration are impeded; and a friction sound can usually be detected by auscultation.

2. *Of the Diffuse Hepatitis.*—The symptoms resemble those of cirrhosis, but a primary enlargement of the liver may possibly occur.

3. *Of Gummata.*—The symptoms are similar to those produced by multiple tumors. The gummata are usually situated near the suspensory ligament and on the under surface of the liver, so that an irregular lower border may be appreciated. Inequalities of the surface may be detected.

The **diagnosis** of syphilitic disease of the liver is made by the consideration of the following points: 1. The absence of alcoholic history. 2. The presence of congenital or the late forms of acquired syphilis. 3. The patient is frequently a young child. 4. The symptoms of cirrhosis are complicated by those of perihepatitis and of gummata. 5. The beneficial results of treatment.

Treatment consists in the administration of potassium iodide in large doses, preferably combined with mercurials. If the treatment be prompt and vigorous, the results are often brilliant; but if the treatment be begun too late, cicatricial bands and diffuse hepatitis may remain, for which further treatment is of no avail. In other respects the treatment of the case is that of ordinary cirrhosis.

ABSCESS OF THE LIVER (SUPPURATIVE HEPATITIS).

Etiology.—Abscess of the liver is due to infection by micro-organisms capable of causing suppuration. Of these micro-organisms the pyogenic micrococci and the amœba coli are the most important. The infecting germ may enter the liver by several channels:

1. *Through traumatism*, by injuries, by foreign bodies, and by parasites. Suppurating hydatid cysts are not uncommon.

2. *Through the portal vein.* Liver-abscess thus compli-

cates intestinal ulcerations (especially those of dysentery, in which the infective agent is the amœba coli), abdominal abscesses, operations upon the rectum, and suppurative inflammations of the portal vein itself.

3. *Through the umbilical vein* in new-born children, following infection of the navel.

4. *Through the hepatic artery* in pyæmia, in malignant endocarditis, and in gangrene of the lung.

5. *Through the bile-duct.* Infection through this channel is favored by the presence of cholangitis or of gall-stones.

6. In some cases no cause can be ascribed for the infection, and to these cases the names " idiopathic " and " tropical " abscess are given. This variety of abscess, which is most common in India, may occur in the Southern United States, but it is infrequent in Northern cities. The infecting agent in most of these abscesses is the amœba coli, although the symptoms of dysentery may be absent.

Abscess of the liver occurs with greatest frequency in men, and in those of adult years.

Pathology.—The wall of the abscess is usually irregular and jagged; it is composed of necrotic liver-tissue infiltrated with fibroid serum and pus, and the surrounding liver-tissue is hyperæmic. In protracted cases a connective-tissue capsule may be formed. The pus may be thick and creamy, or it may be thin, curdy, and of a reddish or brownish color, from pigment derived from blood-cells and broken-down liver-parenchyma, thus resembling anchovy sauce in appearance. In other cases the pus is yellowish-green or brick-red in color, from the staining of bile or of bilirubin-crystals. The pus, which is usually foul-smelling, is generally shown by bacterial examination to be sterile. There may be but one abscess, as in the tropical variety, or the abscesses may be multiple, as is the case with pyæmia and with suppurative inflammation of the portal vein or of the bile-passages.

Single abscesses may reach the size of a cocoanut; multiple abscesses vary in size up to that of a walnut, but by their coalescence still larger abscess-cavities may be formed. Seven-tenths of all abscesses of the liver occur in the right lobe, usually toward the convexity.

Abscesses in the liver evince a tendency to approach the surface and to rupture. In 5 per cent. of all cases rupture into the peritoneal cavity occurs. Usually, however, the peritoneum is shut off by adhesions, so that the abscess ruptures into neighboring organs. Rupture upward into the right lung occurs in 9 per cent., into the right pleura in 5 per cent., of all cases. Perforation of the pericardium or of the vena cava may occur. Rupture downward may occur into the liver, the intestine (3 per cent.), or the kidney. The abscess may approach the chest-wall or the abdominal wall or may open into the gall-bladder. In rare cases the abscess remains stationary. A connective-tissue capsule is ultimately formed, enclosing the purulent contents, which may become inspissated and cheesy.

The **symptoms** are general and local.

General symptoms indicate the presence of pus within the body. Chills are common at the onset, and may be repeated throughout the disease. The chills may be erratic and may be followed by fluctuations in temperature and by cold sweating—a symptom-complex of pus-infection; or the chills may be repeated in multiple abscesses whenever a new focus of suppuration forms. The fever is usually irregularly remittent, although in some cases the remissions are so periodic as to suggest malarial fever. In some cases, especially if long-protracted, fever may be entirely absent. Prostration is evident from the first; the pulse becomes rapid and feeble, and the patient finally passes into a typhoid condition. The appearance of the patient is somewhat characteristic. The skin is sallow and of a slightly jaundiced hue, and the expression strongly suggests the existence of abscess. Marked jaundice is rare. The bowels may be constipated, or there may be diarrhœa. Amœbæ coli may be found in the stools, giving a clue to the diagnosis. The general symptoms may be so complicated or masked by those of the primary disease that no special features may be added to the case.

Local symptoms, due to localized suppuration, consist of pain and tenderness. The pain may be dull in case of a central abscess, or sharp and cutting if the peritoneum over the abscess become inflamed. In many cases the inflammation extends from the peritoneum to involve the diaphragmatic pleura, so that a true pleuritic pain occurs. Pain of a peculiarly heavy, dragging character usually develops whenever the patient lies upon the left side. Tenderness becomes more marked when the peritoneum over the liver is involved. In deep-seated abscess there may only be some rigidity of the abdominal wall in the hypochondrium. In some cases pain and tenderness are slight or are altogether absent.

Portal obstruction occurs only if the abscess happen to press upon the portal vein, but the symptoms are never extreme.

A catarrhal inflammation of the bile-ducts with jaundice may or may not be present, but the jaundice rarely becomes marked.

Physical Examination.—1. *Of the Large Single Abscess.*—The liver is tender on palpation and is irregularly enlarged. There may be appreciable bulging over the abscess, depending upon its size and its position. A large superficial abscess may give rise to fluctuation if the peritoneal surfaces over the abscess be adherent. As the abscess is usually in the right lobe near the convex surface, the area of liver-dulness rises in the axilla, frequently to the fifth rib, and extends across the back on a level with the angle of the scapula. In these cases an erroneous diagnosis of empyema is frequently made. Large abscesses of the upper surface of the right lobe, extending forward, may give rise to dulness as high as the second rib in front and the spine of the scapula behind, while the liver itself is displaced downward, the lower edge being frequently as low as the level of the umbilicus. Abscesses of the right lobe tend to point in the seventh and eighth spaces in the mammary line or below the costal arch. Abscesses of the left lobe usually point in the median line just below the ensiform cartilage.

2. *Multiple Abscess of Pyæmic Origin.*—The liver is usually symmetrically enlarged and tender. Pointing, fluctuation, and bulging are not observed.

In cases of doubtful diagnosis an exploratory aspiration of the liver may be resorted to without risk if strict asepsis be observed. A long needle, not too small in calibre, should be used, and should be deeply inserted in the location of the suspected abscess. The aspiration should preferably be done under ether.

Course of Abscess of the Liver.—1. Some abscesses run a latent course until rupture occurs. In other cases there are found at autopsies old encysted abscesses which gave no definite symptoms during the life of the patient.

2. In some cases the constitutional symptoms are marked, but the local symptoms are slight or are altogether absent. These cases are often mistaken for typhoid fever, malaria, or tuberculosis.

3. The course may be straightforward, with pronounced general and local symptoms. The patient dies from perforation or from septicæmia unless the abscess be opened and drained.

4. The abscess may open into the right lung. At the

time of rupture there will be raised an expectoration of a reddish-brown or brick-dust color, resembling anchovy sauce (Osler), in which amœbæ coli may be found.

5. Chronic abscesses may run an insidious course, with hectic, loss of flesh and of strength, and slight localized pain and tenderness. Amyloid changes in the spleen and the kidneys are liable to occur.

6. In cases of multiple abscesses occurring in the course of pyæmia or malignant endocarditis, pain, tenderness, and moderate enlargement of the liver may be the only additional symptoms.

Diagnosis.—The evident causation of the abscess and the character of the pus obtained by aspiration are of prime importance in diagnosis. Abscess of the liver may be confounded with the following conditions:

1. *Malarial fever.* Here the plasmodium is found in the blood, the spleen is enlarged, and the disease yields to quinine. Pain and tenderness over the liver, if present, are but slight.

2. *Empyema* is to be known by a consideration of its cause and by the different appearance of the aspirated pus. The heart is displaced, and attendant diseases of the lung are evident. It must be remembered that abscess of the liver may rupture into the pleural cavity and constitute a genuine empyema.

3. *Intermittent hepatic fever with gall-stones.* Here the clinical picture may closely resemble abscess; but pain is more sudden and intense, jaundice is marked, and the fever is shorter in duration and is interrupted by periods of apyrexia. Septic symptoms and failure in general nutrition do not occur.

4. *Abscess of the abdominal wall* is diagnosed by the inconsiderable depth of the abscess upon operation.

5. *Subphrenic abscess* is diagnosed by its etiology (rupture

of an ulcer of the stomach or of the duodenum, etc.) and by the frequent addition of air or gas to the purulent effusion.

The **prognosis** is bad unless the pus be evacuated spontaneously or by operation. Multiple small abscesses are usually fatal. Single abscesses treated surgically show a mortality rate of only 30 per cent. Perforation into the lung or into the gastro-intestinal tract is followed by recovery in about half the cases.

The **treatment** is essentially surgical, consisting in the opening and draining of the abscess-cavity. Operative interference is not usually justifiable in cases of multiple pyæmic abscesses, and the operation may be postponed if perforation into the lung or into the gastro-intestinal tract occur, with good drainage, as in these instances spontaneous cure may follow.

TUBERCULAR DISEASE OF THE LIVER.

1. Miliary tubercles in the liver occur almost constantly with miliary tuberculosis, but they give rise to no symptoms.

2. Large tubercular deposits and cheesy masses may occur, usually in connection with tubercular disease of the intestines or of the mesenteric glands, but they are chiefly of pathological interest.

3. In the chronic forms of tubercle in the liver there may be an increase of connective tissue. This "tubercular cirrhosis," as it is sometimes called, occurs especially with chronic tubercular peritonitis and perihepatitis, and gives rise to the symptoms of portal obstruction.

NEW GROWTHS OF THE LIVER.

1. **Cavernous angioma** is not uncommon, but usually is of no practical significance. Exceptionally, in children, angioma may form a growth of considerable size.

2. **Adenoma** may develop; it runs a benign course,

although in some instances it seems to merge into carcinoma.

3. **Leukæmic deposits** will be considered in the description of Leukæmia.

4. **Sarcoma** may occur. This affection is usually secondary, the skin, the eye, and the bones being the favorite seats of the primary growth. Melanotic sarcoma is not infrequent. Sarcoma usually develops in small nodules generally distributed throughout the liver, and the clinical course is that of cancer.

5. **Cancer** of the liver may be primary or secondary. The primary form, which is exceedingly rare, arises from the epithelium of the bile-ducts. It is three times as common in women as in men, and it is usually associated with biliary calculi. Secondary cancer in the liver may follow—(1) any cancer in the distribution of the portal vein, especially in the rectum and the stomach; (2) direct extension from cancer of the pylorus, colon, pancreas, or omentum; (3) more rarely secondary involvement of the liver complicates cancer in other organs at a distance.

Cancer of the liver is more frequent in women than in men, and is more common in advanced life.

Pathology.—There may be one large growth surrounded by smaller deposits, or small nodules may be more generally distributed throughout the liver. These nodules may be sharply defined or may fade imperceptibly into the surrounding liver-tissue. Growths near the surface may be flush with it or may project as round or flattened nodules, at times presenting a central crater-like depression, the so-called "umbilicated appearance." Hemorrhages may occur into the nodules, and there is a tendency to fatty degeneration of the cancer-cells. There is an infiltrating form in which small cancer-masses, varying in size from 3 to 10 millimeters in diameter, are scattered throughout the liver, surrounded by fibrous tissue. The appearance of such a liver closely resembles that of cirrhosis. Symptoms of portal obstruction may or may not be present, according to whether or not the growths happen to press on the portal vein. Large or multiple growths may thus exist without the slightest symptom of portal pressure. In a little less than half the cases there is associated a catarrhal inflammation of the bile-ducts, with jaundice.

Symptoms.—In cases in which the secondary growths are small neither mechanical nor functional symptoms are presented, and the disease runs a latent course. This clinical form occurs especially in cases in which the symptoms of the primary growth are marked. Should the disease run a more classical course, the symptoms of the cancer in the liver are preceded usually by those of the primary growth. When secondary deposits occur in the liver, the primary cancer, especially if it be in the intestines or in the rectum, is apt to cease developing and may be overlooked. Many so-called "primary" cancers of the liver are found to be secondary to a small quiescent cancerous nodule in the rectum.

The symptoms of cancer of the liver may be classified as follows:

1. There are present the symptoms of a *functional disturbance of the liver.*

2. *Symptoms of the cancer itself* are local and general.

Local symptoms consist of pain and tenderness. The pain is usually steady and severe, with paroxysms running through the liver to the back, and at times to the right shoulder. Tenderness is usually marked.

The *general symptoms* are those of cancerous cachexia. The temperature shows a slight elevation in some cases to 100° or 102° F., and may be intermittent with rigors. There is usually some œdema of the feet, the face is of a waxy pallor, and loss of flesh and of strength becomes progressive. Should the local symptoms be slight, the general cachexia may be mistaken for that of anæmia or of nephritis. There is usually gastric disturbance, with loss of appetite, nausea, and vomiting. The cachectic condition rapidly becomes marked, as the patient has usually suffered for some time from the primary growth, and in some cases the cachexia may even precede the enlargement of the liver.

3. *Symptoms of portal obstruction* may or may not be present, as previously explained. Ascites is uncommon, but it may result either from portal obstruction or from cancerous disease of the peritoneum. An enlargement of the spleen occurs in about half the cases.

4. *Jaundice* is present in less than half the cases. The symptom is rarely severe, except in occasional acute attacks. The beeswax appearance of the skin may resemble jaundice, but the conjunctivæ and the urine are not discolored.

Physical Examination.—The liver is regularly enlarged, especially in cases of multiple growths, and in extreme cases may extend from the third rib to the iliac spines. Usually, however, the enlargement does not extend below the umbilicus. The margin of the liver is irregular and nodular, and the contour of the lower border can usually be distinctly appreciated by palpation. The nodular edge moves during inspiration and is continuous with the liver-dulness, thus distinguishing it from the tumor-like feeling of a rolled-up omentum or from growths or cysts of the pancreas. Growths of the pancreas may, however, become adherent to the liver, so that a positive diagnosis cannot be made in every case. The nodules upon the surface of the liver may often be felt through the abdominal wall, and in clear-cut cases the central depression may be appreciated. The infiltrating form—"cancer with cirrhosis" (Osler)—may lead to much enlargement of the liver, and the outlines may be symmetrical. Tenderness is rarely absent, and is of much diagnostic value. The spleen is enlarged in half the cases, and there may be the physical signs of a slight amount of ascites. Important aid in diagnosis is afforded by the detection of the primary growth. Primary cancers of the stomach or the œsophagus usually make themselves evident, but primary growths in the rectum and in the intestines may easily be overlooked. A rectal examination should be made in every case of an enlarged and tender liver in which the diagnosis is obscure.

The **duration** of cancer of the liver is rarely over one year; it may even be shorter than this, as the liver may be involved late in the course of primary visceral cancer elsewhere. As a practical rule, cancer may be excluded if a patient with an enlarged liver be in fair condition at the end of a year.

The **prognosis** is invariably fatal.

Treatment is symptomatic. Opium may be given freely to relieve the pain.

HYDATID OF THE LIVER.

Etiology and Synonym.—The echinococcus is the larval stage of the tapeworm, tænia echinococcus, found in the intestine of the dog and the wolf. The adult worm, which is 4 or 5 millimeters in length, consists of a head with four suckers, a double row of hooklets, and four links; the last link, being alone mature and containing the eggs, is thrown off in the dejecta, and contaminates the food or the drink taken by man. The disease is very common in Iceland, where dogs are allowed great household privileges, one-seventh of all deaths in that country being due to hydatid disease. In Australia and in Europe the disease is not uncommon, but it is exceedingly rare in America. *Synonym:* Echinococcus cyst.

Pathology.—Entering the intestine by contaminated water or food, the embryo is carried by the portal vein to the liver, where it may lodge; or it may pass through the liver, enter the vena cava, and become lodged in more distant parts. In the liver it grows to form a cyst which may be unilocular or multilocular. The cyst-wall is composed of two distinctive layers—an outer layer, of a fine, structureless, laminated membrane peculiar to hydatid cyst, and an internal granular layer, the "endocyst." In time, surrounding the cyst there is gradually developed a capsule of connective tissue. The cyst is filled with a clear, non-albuminous fluid containing sodium chloride and a trace of sugar; the specific gravity of this fluid varies between 1005 and 1015. After the cyst has grown for from four to six months and has attained the size of a walnut, there develop from the inner granular layer little pediculated vesicles, the walls of which are identical with those of the primary cyst, and which are known as "brood-capsules." These brood-capsules contain a number of echinococcus heads or "scolices." Each scolex, consisting of a head, suckers, and hooklets, may project into the cavity of the brood-capsule or may retract or invaginate itself so as to form a prominence upon the outer side of the capsule. Cysts may form within the parent-cysts (daughter-cysts), and smaller cysts (granddaughter-cysts) may develop within these. Some of the cysts may be sterile, containing no scolices. The daughter- and granddaughter-cysts may remain connected with the ancestral cyst-wall, but in time they tend to free themselves. Free hooklets are often found; they are of absolute diagnostic value when detected in the fluid obtained by an exploratory aspiration.

In the multilocular form of hydatid disease the secondary cysts are surrounded with fibrous tissue, so that there is formed a large irregular tumor of connective tissue arranged in septa, enclosing spaces in which are found the cysts;

these spaces may also contain gelatinous material. The cysts are usually sterile. The peculiar characteristics of the multilocular form are attributed to the growth of the brood-capsules outward (exogenous cysts) into the tissues of the liver.

The growth of hydatid cysts is exceedingly slow, extending over many years. In some cases spontaneous cure occurs—a connective tissue capsule is formed, the cyst-wall becomes calcified, and the contents become putty-like and may contain free hooklets. In other cases rupture may occur in any direction. In a few cases the cyst suppurates and assumes the character of an abscess of the liver.

Symptoms.—In the majority of cases the symptoms are purely mechanical, due to the enormous enlargement of the liver, and consist of a sense of weight and dragging and of dyspnœa. There may be pressure on the portal vein, with symptoms of its obstruction—enlargement of the spleen, gastro-intestinal disturbances, and ascites. Pressure on the bile-ducts causes obstructive jaundice. Pressure on the vena cava may result in œdema of the legs. Suppuration of the cysts is attended by the symptoms of pyæmia and of abscess of the liver. In the multilocular variety portal obstruction and jaundice are not uncommonly observed. Rupture into the pericardium, the pleura, the peritoneum, the gall-bladder, or the vena cava is usually attended by a fatal result. Rupture into the lung may allow of partial drainage of the cyst, or may be complicated by pneumonia, abscess, or gangrene of the lung. Recovery may also follow rupture into the alimentary tract or through the abdominal wall.

Physical Examination.—There is usually considerable bulging in the epigastrium or in the hypochondrium. The liver is much increased in size, so that it may extend from the second rib to the pelvis. The enlargement, however, is not uniform, so that the outline of the liver is exceedingly irregular. Deep-seated cysts give rise to an elastic feeling on palpation; superficial cysts give rise to fluctuation. Usually the surface of the tumor is smooth and free from all irregularities. An "hydatid fremitus," resembling the quivering of jelly, may be detected. According to Murchison, this fremitus is not due to the striking of a daughter-cyst against the parent-wall, as it may be detected in barren hydatids, but may be elicited over any large cyst with thin, tense walls and fluid contents; however, as the only cysts occurring in the liver with these characteristics are hydatids, the fremitus is of much diagnostic value. Unfortunately, the sign is not always present. Exploratory aspiration renders the diagnosis positive from the character of the watery contents, by the presence of free hooklets, and by the occasional presence of small rolled-up bits of the outer laminated membrane.

The **prognosis** is rendered grave by the possible complications that may ensue.

Treatment is entirely surgical. The older methods of treatment by aspiration and the injection of iodine have been abandoned altogether.

FATTY LIVER.

Two varieties of fatty liver are recognized—fatty infiltration, in which the fat is derived from some constituent of the food, and fatty degeneration, in which the fat is due to changes occurring within the liver-cell. The latter form occurs with phosphorus-poisoning, yellow fever, and acute yellow atrophy of the liver.

Etiology.—Fatty liver may be physiological after a hearty meal and during lactation. The disease occurs with habitual over-eating, especially of fatty and starchy food, with intemperance (frequently associated with cirrhotic changes), and with indolent and sedentary habits, especially in those who over-eat. It is common with cachectic conditions and wasting diseases in which the oxidation-processes are defective. Thus it frequently occurs with phthisis and with profound anæmias, and less commonly with rickets. The disease occurs at all ages, and is more common in females than in males.

Pathology.—The liver is enlarged to such an extent that its lower border usually reaches the umbilicus; it may even be larger than this. The margins are rounded, and the organ shows a greasy appearance. The specific gravity of the liver may be so low that the organ will float in water. Cut sections are anæmic and of a yellowish color, and the knife is smeared with fat; the stroma is normal. The lesion may be complicated by catarrhal inflammation of the bile-ducts.

Symptoms.—The disease may run a course without symptoms, or there may be disturbances in the functional activity of the liver, with a sense of weight and fulness in the hypochondrium. In some cases there is pain when the patient lies upon the left side, from the mechanical stretching of the peritoneal ligaments. Portal obstruction, marked jaundice, and pain and tenderness are absent in this disease. In exceptional cases, however, fatty liver does not run the benign course usually ascribed to it, but becomes a formidable disease.

1. In some cases the symptoms of functional disturbance are marked, symptoms of fatty heart are present, and there is a chronic gastritis, possibly with attacks of hæmatemesis. The patient becomes emaciated and dies exhausted. These cases somewhat resemble cancer of the liver, but are unaccompanied by pain.

2. Other patients at some time develop acute gastritis of a severe type, with cerebral symptoms, weak heart, and possibly hæmatemesis. These attacks of gastritis are frequently fatal.

3. Symptoms of a malignant jaundice may develop, although the autopsy fails to reveal obstruction of the bile-ducts. These attacks of jaundice resemble those occurring in the latter stages of biliary cirrhosis, and are usually fatal.

Physical Examination.—The liver is symmetrically enlarged, but it rarely reaches below the umbilicus, as does the waxy liver. The organ is soft and smooth on palpation, and the lower edge is distinctly rounded. There is neither ascites nor enlarged spleen. Examination is frequently unsatisfactory because of the obesity of the patient.

The **prognosis** is bad for recovery, but the patient may live for years. The fatty liver of wasting diseases may hasten the fatal issue by interfering with the patient's nutrition. The occurrence of fatal gastritis or jaundice must always be considered in giving a prognosis.

The **treatment** is that of the original cause. Exercise is to be regulated and enforced in those indolently inclined; the use of fat, sugar, and starch should be restricted, and a diet of lean meat, green vegetables, and milk is to be recommended. Indulgence in alcoholic or malt liquors is to be prohibited. The medicinal treatment is that of functional disturbance of the liver. In phthisical patients the condition of the liver should not be treated, but superalimentation, especially by means of fat, cream, and cod-liver oil, should be continued notwithstanding the effect of such a diet upon the fatty infiltration of the liver.

AMYLOID LIVER.

Etiology and Synonyms.—This condition arises from long-continued suppuration, especially of the lungs or the bones. It occurs with constitutional syphilis, it may follow rickets or prolonged convalescence from infectious fevers, and it may complicate cancerous cachexia. *Synonyms:* Waxy liver; Lardaceous liver.

Pathology.—The liver is symmetrically enlarged, the lower border being frequently found below the navel. The organ is firm and hard; its lower edge is sharp. The cut section is anæmic, and in places shows a translucent appearance resembling raw bacon. The amyloid areas are stained mahogany-brown by dilute watery solutions of iodine. The amyloid change begins first in the small blood-vessels and extends to the connective-tissue stroma. The liver-cells do not become waxy, but they may be atrophied by pressure or may undergo fatty degeneration. Similar waxy changes are found in other viscera, especially in the spleen and the kidney.

Symptoms.—There are no characteristic symptoms. Jaundice does not occur. There is no portal obstruction, unless the portal vein be compressed by an enlarged waxy gland in the hilum of the liver.

Physical examination reveals a symmetrical painless enlargement of the liver. As the patient is usually emaciated, the detection of the enlargement is never difficult, and the sharp lower edge may be appreciated distinctly. The spleen is enlarged, and the waxy condition of the kidney is made evident by the passage of an increased amount of urine containing albumin, and generally by œdema of the lower extremities.

Prognosis and **treatment** are those of the causative disease.

JAUNDICE (ICTERUS).

Etiology.—Two varieties of jaundice are described. The first, the obstructive or *hepatogenous* jaundice, is due to obstruction of the bile-ducts and results from the absorption of the bile-pigments. The second or *hæmatogenous* variety includes those cases of jaundice in which no obstruction exists. The causes of jaundice are thus classified:

1. *Obstructive or Hepatogenous Jaundice.*—(*a*) *Foreign bodies* within the duct—gall-stones, inspissated bile, parasites, and bodies entering from the duodenum.

(*b*) *Catarrhal inflammation* of the bile-passages or of the duodenum.

(*c*) *Stricture or obliteration of the common duct*, congenital and cicatricial.

(*d*) *Pressure on the duct from without*—(1) from enlarged glands in the hilum of the liver; (2) from neighboring tumor, as of the liver, stomach, or pancreas; (3) from large abdominal tumors, as aneurysm of the abdominal aorta, fecal impaction, ovarian tumor, and pregnancy; (4) from contracting peritoneal bands and thickening; (5) from lowering of the blood-pressure in the liver so that it is less than the pressure of the bile within the smaller ducts, as in mental shocks and cerebral concussion.

2. *Non-obstructive or Hæmatogenous Jaundice.*—The following classification is given by Osler:

(*a*) From widespread necrosis of liver-cells, interfering with their bile-forming function, as in acute yellow atrophy and in certain cases of hypertrophic cirrhosis.

(*b*) The toxic form. The poisons of yellow fever, Weil's disease, malaria, typhoid fever, epidemic jaundice, pyæmia, snake-bites, chloroform, ether, phosphorus, and mercury cause such an increased destruction of the blood-cells (hæmolysis) that more blood-pigment is set free than can be eliminated, and is retained in the tissues.

Symptoms.—1. *Of Hepatogenous Jaundice.*—The jaundice first appears in the conjunctivæ and the urine, and then appears in the skin and the mucous membranes. The color of the skin varies from a canary-yellow to an orange-green or even to a greenish-black. The constant tinting of the conjunctivæ and the presence of bile-pigment in the urine distinguish jaundice from all other pigmentations or stainings of the skin. The urine, which is deeply bile-stained and is often of a brownish color resembling porter, foams readily, and the froth is yellow; it stains linen and reacts to Gmelin's test. The abundant yellow foam distinguishes the urine of jaundice from urine discolored by the internal use of rhubarb or of santonin. Hyaline tube-casts are not uncommon, and in severe or protracted cases there may be a moderate amount of albumin in the urine. The perspiration is bile-stained and will stain linen, but the saliva, the tears, and the milk are not discolored. Pruritus of the skin is severe and distressing, especially in protracted cases. Digestive disturbances arise from the fact that no bile passes into the intestine. The assimilation of fats is interfered with, so that the patient loses flesh and becomes anæmic. There is a bitter taste in the mouth; the tongue is heavily furred. Abdominal flatulence develops. The bowels are usually constipated, although diarrhœa may occur if fermentative changes take place within the intestine. The stools are clay-colored, pasty, or putty-like, and are usually offensive. The heart-action is slower and more forcible than normal, in severe cases falling to 40, or even to 20, in the minute. The slowness of the pulse is best observed when the patient is resting, as the heart-action may become rapid and feeble upon exertion. There is no fever with simple jaundice; on the contrary, the temperature is more apt to be subnormal —96° or 97° F. In severe or protracted cases there is developed a tendency toward hemorrhage. Purpuric spots and ecchymoses appear beneath the skin and the mucous membranes, and hemorrhages from free mucous membranes may occur, especially from the nose, the stomach, and the intestines. The hemorrhagic tendency may be so extreme as to preclude surgical treatment, as in operations the bleeding from the incision may become uncontrollable.

Cerebral symptoms are almost constant. In mild cases there are persistent headache, irritability of temper, and utter inability to concentrate the mind. Severe cerebral symptoms resemble those occurring with uræmia, and comprise delirium, convulsions, stupor, and coma; or the patient may pass into the typhoid condition with hemorrhages. The temperature in these severe cases may be as low as 87° F., but a high ante-mortem rise to 106° or 108° F. is commonly observed. To these severe cases with cerebral symptoms the names "pernicious jaundice," "cholæmia," "acholia," and "cholesteræmia" have been given. The best explanation of these cases is given by Frerichs, who ascribes the toxæmia to the pernicious influence of unknown substances which, under normal conditions, should be elaborated by the liver into bile.

2. *Of Hæmatogenous Jaundice.*—The symptoms are often obscured by those of the primary disease. As a rule, the tint of the skin is lemon-colored. The urinary pigments are usually increased, but, as a rule, bile-pigment is not found in the urine. As bile enters the intestine, the stools do not become clay-colored. The pulse is not generally lowered in frequency, but in the toxic form severe cerebral symptoms are apt to develop.

The **treatment** of jaundice as a symptom will be given under the heading of Catarrhal Jaundice.

ACUTE FEBRILE JAUNDICE.

(See *Weil's Disease.*)

CATARRHAL JAUNDICE.

Etiology and Synonym.—This most common disorder is due to the closure of the terminal end of the common bile-duct by swelling or by a plug of mucus during the course of an acute gastro-duodenitis. The inflammation in some cases extends upward along the common bile-duct. The gastro-duodenitis is usually due to some dietetic error. It may occur at any age, but it is especially frequent in young people. The occurrence of the duodenal catarrh is favored by portal obstruction and by chronic heart disease. Such is the low tension of the bile within the duct that a very moderate amount of swelling or occlusion prevents the bile from entering the intestine and results in obstructive jaundice. *Synonym:* Gastro-duodenitis.

The **symptoms** are those of gastritis and of jaundice. The gastric symptoms are the first to appear, and begin insidiously with malaise, loss of appetite, nausea, and vomiting. The bowels are usually constipated. There may be epigastric pain and tenderness. Mild cases are unaccompanied by fever, but in the more severe forms there may be a temperature of from 100° to 102° F. After the gastric symptoms have lasted for a few days jaundice appears, but the deeper greenish-yellow tints are not seen. The regular symptoms of jaundice are present, as described in the preceding article. After about a week the patient begins to improve and the gastric symptoms become less marked. The jaundice is slower in disappearing, so that the whole duration of the disease is usually from four to eight weeks. There are mild cases in which jaundice may be the first, and even the only, symptom, and the jaundice may terminate within two weeks.

The **diagnosis** is usually made with ease. Doubt should be attached, however, to those cases in which the jaundice persists for over three months.

The **prognosis** is good. In exceptional cases gastro-duodenitis with jaundice may occur in a patient who has a fatty or cirrhotic liver, and may terminate fatally.

Treatment.—No very active treatment is indicated in the mild cases. The patient is better for keeping about, in spite of his prominent color. The treatment is in the main that of an acute gastric catarrh. Cholagogues are of great service, provided that by their administration the gastritis is not aggravated. Of the cholagogues, the best is sodium phosphate in ʒj doses before meals. Benzoic acid, sodium benzoate, sodium salicylate, small doses of rhubarb, and an occasional mercurial purge are also recommended. Sodium bicarbonate may be given in ʒj doses, in Vichy water, between meals; or any of the simple alkaline mineral waters may be given, of which Vichy is perhaps the best. Rectal injections of cold water certainly do good in diminishing the jaundice. From 1 to 2 quarts of water should be slowly injected into the rectum once a day, and retained as long as possible. At first the temperature of the water should be 60° F., but in later injections the temperature of the water may be reduced gradually, so that finally ice-water may be used. Pruritus is to be treated by bathing the itching parts with a solution of carbolic acid (1 : 200) or one of bichloride of mercury (gr. j : ℥j); or a saturated solution of sodium bicarbonate may be used. In some cases a calomel ointment affords relief. In severe cases internal remedies may be necessary, as phenacetine or sodium bromide.

CHOLELITHIASIS.

Etiology and Synonyms.—Gall-stones occur more frequently in women (75 per cent. of cases) than in men, and especially in women between thirty and sixty years of age. Tight lacing, over-eating, especially of saccharine and starchy food, and sedentary modes of life seem to act as important factors in their causation. The patients are usually stout, and the combination of fatty liver with biliary calculi is exceedingly common. Of women with gall-stones, 90 per cent. have borne children. *Synonyms*: Gall-stones; Biliary calculi.

Pathology.—Biliary calculi are in almost all instances formed within the gall-bladder, but in rare cases they may arise in the small ducts within the liver. Stones found in the larger ducts do not originate there, but become impacted on their way to the intestine along the duct. Two varieties of biliary calculi are found, one consisting of inspissated bile, and the second comprising calculi proper.

1. Inspissated bile occurs as a brown or greenish-black semi-solid mass composed of the solid constituents of bile with the admixture of mucus. The same symptoms may arise from inspissated bile as from the regular calculi.

2. The regular biliary calculi are not always composed of the same ingredients.

(*a*) Some are composed of cholesterin, alone or with bile-pigment.

(*b*) Some are composed of cholesterin, bile-pigment, and the salts of lime and magnesium. These two are the ordinary varieties. The calculi are soft, light and can be cut easily with a knife.

(*c*) Rarely the calculus consists of bile-pigment alone or of calcium carbonate alone. These latter varieties form small hard gravel, the so-called "gall-sand." There is usually a central nucleus, which may consist of mucus or of bile-pigment.

The number of calculi that may be found in the gall-bladder varies up to several thousand. The calculi vary in size from a grain of sand to that of a lemon; the greater their number the smaller they are. The color varies from white to a waxy lemon or a greenish-yellow, according to the amount of pigment they contain. More rarely they are bronze-green or even black. The purer cholesterin-calculi are nearly white, are of a crystallized structure, and readily cut like wax. The calculi are round, irregular, or oblong, or they may be polyhedral and faceted by mutual pressure and friction.

The exact reason for the formation of biliary calculi is unknown. It is a fact that cholesterin is precipitated if the bile becomes acid, but whether this precipitation actually occurs within the body is unknown. It is supposed that cholesterin is also precipitated whenever there exist defects in the sodium salts. It is known that the formation of calculi is favored by any cause retarding the flow of bile and by all unhealthy conditions of the bile-passages.

Symptoms.—Gall-stones give rise to symptoms in three different ways:

1. By their passage through the ducts; 2. By their impaction in the ducts; 3. By their retention within the gall-bladder.

1. *Passage through the Ducts.*—Small stones may pass from the gall-bladder through the cystic and common ducts without symptoms, or there may result slight jaundice and discomfort in the region of the liver. Larger calculi engaging in the ducts give rise to the symptoms of biliary or hepatic colic—pain, vomiting, fever, jaundice, and slight enlargement of the liver. The attack is sudden, usually occurring several hours after a heavy meal or after muscular exertion. The pain starts from the epigastrium and radiates through to the back and upward, so that it may be felt in the right shoulder. Radiation of the pain downward is exceedingly rare—a point of differential diagnosis from renal colic. There are two elements to the pain—a steady grinding ache, with sharp cutting or stabbing paroxysms of unbearable intensity. During the paroxysms of pain the face is pale, the body is bathed in perspiration, and attacks of syncope and convulsions are not infrequent. Fatal syncope or collapse has occasionally been observed. At the onset the pain may be relieved by pressure, but later tenderness develops, especially over the gall-bladder. Nausea and vomiting almost regularly occur during the attack.

The vomited matters contain no bile if the common duct be obstructed, but in the vomita biliary calculi may be found. Chills are common at the onset of the attack, and recur at intervals which, in protracted cases, may be of such regularity as to resemble malarial fever. The temperature is temporarily raised during the attack to 102° or 104° F., but if the attack be slight and of short duration there may be no fever. In protracted cases, especially if there be a complicating inflammation of the biliary passages, there may be an intermittent or remittent fever resembling that of malaria and accompanied by repeated rigors. This "intermittent hepatic fever" also occurs with impaction of the stone within the common bile-duct.

The pulse during an attack of biliary colic is often slow, but it may be rapid, feeble, or irregular. In from twelve to twenty-four hours jaundice appears, with the regular symptoms of the obstructive variety, but it usually disappears in about a week after the close of the attack. Persistence of the jaundice after this time suggests catarrhal inflammation of the bile-ducts (catarrhal cholangitis) or impaction of the calculus within the duct. Jaundice is not, however, constant, as it does not appear in case of a very small calculus within the common bile-duct, not entirely occluding it, or in the case of a calculus passing the cystic duct. Toward the close of the attack the liver is moderately enlarged and tender, and the gall-bladder may be felt to be distended.

The attack terminates suddenly with the dropping of the calculus into the duodenum, and the sharp pain ceases at once, leaving a dull ache which may persist for several days. Sudden cessation of pain is also observed in cases in which a calculus engages in the cystic duct and then drops back again into the gall-bladder.

The *duration* of an attack of biliary colic varies from several hours to days. Protracted cases are usually due to the merging together of a series of attacks. Recurrences are common, because the biliary calculi usually occur in the gall-bladder in great numbers.

The *diagnosis* of cholelithiasis is rendered positive by finding the calculi in the stools or in the vomited matters. The stools should be tied up in a gauze bag, which is to be attached to a water-faucet so that the feculent matter may be washed through the meshes of the gauze, leaving the calculi in the bag. A faceted appearance of a calculus implies that it is one of many.

The *prognosis* for the attack itself is good, although fatal syncope, collapse, and rupture of the duct have occurred. Cerebral hemorrhage has repeatedly been observed during an attack.

2. *Impaction in the Ducts.*—The symptoms of impaction follow those cases of biliary colic in which the pain gradually ceases, leaving a considerable dull aching feeling; they differ according to whether the cystic or the common bile-duct be the seat of the impaction.

(*a*) *Impaction in the Cystic Duct.*—There is usually no jaundice, but the effects of the impaction fall upon the gall-bladder, which may undergo the following changes:

(1) The gall-bladder becomes distended with thin, colorless, mucoid contents—the so-called "dropsy of the gall-bladder," or "hydrops vesicæ felleæ." A large globular tumor is thus formed, projecting from the under surface of the liver to the right of the median line. The distended gall-bladder may reach such dimensions as to extend to the navel or even to the level of the anterior spines. In some cases the gall-bladder becomes distended without projecting beyond the lower border of the liver.

(2) Acute phlegmonous cholecystitis may occur, with perforation and fatal peritonitis, but the condition is one of great rarity.

(3) Suppurative cholecystitis, or empyema of the gall-bladder, is of not infrequent occurrence. The bladder is distended by pus and is the source of pain and tenderness. Septic symptoms are those of a localized abscess. Perforation may occur into the peritoneum, into hollow viscera, or through the abdominal wall, forming biliary fistulæ; or there may be formed an acute circumscribed peritoneal abscess. Calcification of the gall-bladder or lime-incrustations of the mucosa may follow the drainage of the empyema.

(4) Atrophy of the gall-bladder may result in its transformation into a little ball of connective tissue.

(*b*) *Impaction within the common bile-duct* causes persistent obstructive jaundice. The secondary results of such an impaction fall upon the bile-passages and the liver. The gall-bladder is seldom involved.

(1) *Inflammation of the Biliary Passages.*—There may be a *catarrhal cholangitis.* The biliary ducts are dilated and filled with a colorless mucoid fluid. The symptoms are those of jaundice with "intermittent hepatic fever." Chills occur, with elevations in temperature to 103° or 104° F., the fall of the fever being accompanied by sweating. These ague-like paroxysms may occur with such periodicity as to be mistaken for malaria, and they may occur at intervals for months or even for years. With each paroxysm the jaundice deepens, and pain over the liver and gastric disturbances are observed. In the intervals between the paroxysms the patient is deeply jaundiced, but the temperature is normal and there is rarely any depreciation in the general health. The exact cause for this hepatic fever is not known.

(2) *Suppurative cholangitis* is attended by suppuration extending along the biliary passages and frequently involving the gall-bladder. Suppuration may extend to the liver

surrounding the ducts, so that the organ is penetrated by long, branching abscess-cavities. The liver becomes enlarged and tender, symptoms of septicæmia or of pyæmia develop, and the disease runs a short and fatal course.

The effect on the liver of such an impaction in the common duct is to cause its enlargement. The liver is rendered firmer by deposits of connective tissue along the course of the biliary ducts, and the lesions of biliary cirrhosis may thus ultimately develop.

When a calculus becomes impacted in either the cystic or the hepatic duct, it may there remain indefinitely, or it may progress along the duct by starts, so that finally the passage to the duodenum is accomplished. This favorable outcome of the impaction is more common if several calculi are wedged together, so that at any time the wedge may be broken up and the individual calculi be passed without difficulty.

Perforation of the duct is of common occurrence, and biliary fistulæ thus formed may open through the abdominal wall, into the abdominal cavity, into the duodenum or the colon, more rarely into the gall-bladder, the substance of the liver, the lungs, the stomach, the small intestine, or the pelvis of the kidney. The calculi may be discharged through the fistulous opening, frequently in great numbers, and spontaneous cure may result. Obstruction to the bowels may result from the perforation of large solitary gall-stones into the duodenum. In other cases perforation of the duct leads to the formation of a circumscribed peritoneal abscess.

In some instances a calculus may pass into the common duct and remain there without actually obstructing the duct. A slow increase in the size of the calculus is compensated by a gradual dilatation of the duct.

3. *Retention of calculi within the gall-bladder* is of such

common occurrence as to be found in 25 per cent. of all autopsies made on women over sixty years of age. In many cases this condition gives absolutely no symptoms, but post-mortem examination may show the gall-bladder nearly empty of bile and contracted upon the calculi, with obliteration of the cystic duct by a calculus. In the majority of cases, however, calculi from time to time enter the cystic duct and pass to the duodenum or drop back into the gall-bladder. In either case symptoms of biliary colic are observed, but in the latter instance jaundice does not occur and the pain may be of short duration. Calculi remaining in the gall-bladder may excite a catarrhal or suppurative inflammation of its wall. Catarrhal cholecystitis is attended by a globular enlargement of the gall-bladder, with pain and tenderness. The symptoms of empyema of the gall-bladder have already been described.

Treatment of Cholelithiasis.—1. During the attack of biliary colic the indications are to relieve pain and spasm. A hot bath is of comfort, and until the bath can be prepared hot applications over the liver are of service. Hypodermic injections of morphine (gr. $\frac{1}{4}$) with atropine (gr. $\frac{1}{150}$) are to be given, and in severe cases whiffs of chloroform may be necessary until the patient is under the effect of the opiate. It should be remembered that the pain may cease suddenly, and, tolerance for the morphine being thus lessened, toxic effects may result if too large doses have been given. Copious draughts of hot water containing sodium bicarbonate (ʒj : Oj) exert a sedative effect, provided that vomiting is not induced.

2. If the ducts be occluded, various methods have been recommended to dissolve the calculus or to dislodge it mechanically. Olive oil in large doses has been recommended, but it is useless, the alleged calculi passed after its administration having been proved to be lumps of fatty

concretions. Durande's formula, consisting of 3 parts of ether and 2 parts of turpentine, may be given in from 15- to 20-drop doses three times a day. It is much used by European physicians, but is of doubtful utility. Chloroform internally and salicylate of soda have been recommended, but no drug thus far has proved to be of any value in dissolving the stone.

If the impaction be accompanied by inflammatory symptoms, the stone may be removed surgically, or the duct may be exposed and the calculus be broken up without incising the wall of the duct.

3. If empyema of the gall-bladder occur, the condition demands surgical treatment. For cases of repeated attacks of gall-stone colic, with accumulations of calculi in an inflamed gall-bladder, the operation of cholecystotomy, or opening the gall-bladder and removing the stones, has been remarkably successful.

Dropsy of the gall-bladder may be treated by repeated aspirations.

To prevent the formation of gall-stones, highly seasoned and indigestible food should be avoided, and total abstinence from alcohol should be insisted upon. The bowels should be kept freely open, and abundant systematic exercise should be enforced. Hot morning draughts of the alkaline mineral waters are of service, or sodium phosphate in dram doses may be given in a tumblerful of hot water before breakfast.

CANCER OF THE GALL-DUCTS.

Cancer of the liver starting from the endothelium of the biliary passages has already been described.

The common bile-duct in rare instances is the seat of the primary cancer. The symptoms are those of chronic obstructive jaundice and of cancerous cachexia. The diagnosis is seldom, if ever, made during life, and no treatment is available except that for the relief of pain.

Cancer of the gall-bladder is also exceedingly rare. The tumor reaches considerable size, usually that of a child's head.

Secondary nodules in the liver occur by direct extension or by metastasis through the portal vein. The symptoms are those of an enlarged, tender, and painful tumor in the location of the gall-bladder, of secondary growths in the liver, and of cancerous cachexia. The early symptoms resemble those of gall-stones. Treatment is palliative, unless an early diagnosis renders it possible to remove the gall-bladder by operation.

DISEASES OF THE BLOOD-VESSELS OF THE LIVER.

Hepatic Artery.—Aneurysm of the hepatic artery has been observed in a few cases. There may be pressure on the portal vein or on the bile-ducts.

Hepatic Vein.—Affections of the hepatic vein are almost unknown.

Portal Veins.—1. *Chronic portal obstruction* may arise from chronic diseases of the heart and the lungs and from tumors of the mediastinum pressing upon the vena cava. The portal obstruction thus caused is accompanied by œdema of the lower extremities and by congestion of the kidney.

Local causes for portal obstruction are cirrhosis, thrombosis, and pressure on the vein by tumors in the liver or in its vicinity. The vein may be compressed by proliferative peritonitis involving the grastro-hepatic omentum.

The *symptoms* and *treatment* of chronic portal obstruction have been described at length in the consideration of Cirrhosis of the Liver.

2. *Thrombosis; Adhesive Pylephlebitis.*—Primary thrombosis of the portal vein may occur as a terminal event in dying patients, but it is of no practical importance. Secondary thrombosis is regularly due to pressure upon the portal vein or upon one of its branches.

Pathology.—The vein is filled with a recent clot; later the thrombus becomes paler and harder, and may ultimately be converted to connective tissue.

The *symptoms* are those of sudden portal obstruction. The abdominal veins become distended; the spleen is enlarged; vomiting, hæmatemesis, diarrhœa, occasionally with bloody stools, and rapidly forming ascites comprise the prominent symptoms. In no other disease are these symptoms of portal obstruction so rapidly developed.

If the portal vein itself or one of its larger branches be occluded, an efficient collateral circulation is impossible. Death results in a few days from prostration and heart failure, which are added to the above-mentioned symptoms. If the collateral circulation can be established, a certain amount of improvement results, and the patient may live for years with indefinite symptoms of moderate portal stasis; but death finally results from emaciation, from exhaustion, or from hemorrhages.

The *prognosis* of thrombosis of the larger branches of the portal vein is always bad. If a smaller branch be occluded, the prognosis depends upon the establishment and maintenance of satisfactory collateral circulation.

The *treatment* is that of portal obstruction by tapping, diuretics, and cathartics.

3. *Septic Thrombosis; Suppurative Pylephlebitis.*—The thrombus is not due to mechanical pressure, but is secondary to a septic inflammation of the wall of the vein. Infection is caused by intestinal ulceration, by abdominal abscesses, or by sharp foreign bodies, as fish-bones, which

penetrate the wall of the intestine and infect a branch of the portal vein. In new-born children the condition may arise from sepsis entering through the navel and infecting the clot in the umbilical vein.

Pathology.—By the influence of bacteria the clot softens and breaks down to form a puriform mass. Septic emboli, entering the liver, give rise to multiple abscesses, and even to general pyæmia.

The *symptoms* are those (1) of portal obstruction, (2) of multiple abscesses in the liver, and (3) of pyæmia or septicæmia.

Prognosis.—The disease is invariably fatal, usually within two weeks.

The *treatment* is symptomatic. Operative interference is, of course, impossible.

6. DISEASES OF THE PANCREAS.

HEMORRHAGE.

Hemorrhage into the pancreas is of rare occurrence. The subjects have usually been males of adult life. Injuries and alcoholism seem to bear some causative relation to the disease, but the precise causation is unknown.

Pathology.—Hemorrhagic infiltration is found not only in the substance of the pancreas, but also in the subperitoneal tissue in its vicinity.

Symptoms.—There is suddenly developed intense pain, referred to the upper part of the abdomen; nausea, vomiting, and abdominal tenderness rapidly follow. The patient becomes anxious, restless, and passes into a fatal collapse. In the rapidly fatal cases death may result in from half an hour to two hours. In less severe cases the symptoms merge into those of hemorrhagic pancreatitis.

Prognosis.—Theoretically it is possible for recovery to follow small hemorrhages, but practically the disease is almost certainly fatal.

Treatment is directed toward the pain and the collapse.

ACUTE HEMORRHAGIC PANCREATITIS.

Etiology.—The subjects of pancreatitis are usually males in adult life. The majority of the reported cases have been preceded by hemorrhage into the pancreas, by alcoholism, by injury to the abdomen, or by repeated attacks of gastro-duodenitis, but the precise causation of the disease is unknown.

Pathology.—The pancreas is enlarged and is infiltrated with blood. The color of the gland is mottled red and yellow and dead white. The latter color is due to areas of "fat-necrosis" of Balser.[1] The same lesions may also be found in the root of the mesentery, in the mesocolon and the omentum, and in the retroperitoneal tissues in the vicinity of the pancreas. If the patient survive long enough, gangrenous pancreatitis may develop.

The tallow-like appearance of the areas of fat-necrosis is due to the crystallization of fat, as the area is found to consist of stearin, alone or combined with lime-salts. The crystallization is supposed to be due to contact with the pancreatic secretion. In the areas of fat-necrosis various bacteria have been found, the most prominent being the bacillus coli communis.

The **symptoms** begin suddenly with an intensely violent pain, usually referred to the upper abdominal region, but which may be more general than this. Vomiting is frequent and usually incessant. The vomited matters are bilious and may contain blood. The temperature may be normal, subnormal, or slightly elevated. The bowels are usually constipated. The abdomen becomes tympanitic and tender. Collapse-symptoms rapidly supervene, and death usually results from the second to the fourth day. Should the patient survive, the symptoms of gangrenous pancreatitis usually appear.

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[1] Disseminated areas of fat-necrosis may in rare instances be scattered throughout the abdomen, even in cases in which the pancreas is found to be normal.

The **diagnosis** from intestinal obstruction and from perforative peritonitis cannot be made with any certainty.

Prognosis.—The disease is almost certainly fatal, although one case of recovery has been reported by Osler.

The **treatment** is that of the pain and the collapse.

GANGRENOUS PANCREATITIS.

According to Fitz, hemorrhagic pancreatitis not terminating fatally or in improvement within a week usually results in gangrenous pancreatitis. The pancreas is converted to a gangrenous mass lying in a meshwork infiltrated with green offensive fluid; or the sloughing mass may lie in the lesser omental cavity, attached only by a few loose threads to its walls, and may be discharged through the bowels as a slough. In rare cases spontaneous cure has thus resulted. The omental cavity contains an ichorous fluid or pus, and general infection of the peritoneum may occur. Disseminated areas of fat-necrosis are usually present, and there may be suppurative pylephlebitis, pericarditis, or pleurisy.

Symptoms.—The disease begins as a hemorrhagic pancreatitis. Symptoms of peritonitis supervene, usually localized in the upper portion of the abdomen, corresponding to the omental cavity. Collapse precedes the fatal issue.

The **duration** of the disease is from ten to twenty days.

The **prognosis** is bad, with the exception of the very rare cases in which the slough is passed by the bowel.

The **treatment** is that of a circumscribed peritonitis, and is distinctly surgical.

SUPPURATIVE PANCREATITIS.

Etiology.—The method of infection is unknown.

Pathology.—The pancreas may be studded with small abscesses or may be converted to a cyst filled with pus. A diffuse peritonitis may result, or the abscess may rupture into the lesser omental cavity or into the duodenum. Suppurative pylephlebitis and abscesses in the liver are not infrequent. Areas of fat-necrosis are but rarely observed.

Symptoms.—Epigastric pain, vomiting, and prostration mark the onset of the disease. Septic symptoms develop, abdominal distention and tenderness become marked, and death results in from two to four weeks. Other cases run a more chronic course extending over weeks or months. Fever is slight or altogether absent. There is but little pain, although tenderness is elicited in the region of the pancreas. The patient becomes weak and thin, and dies exhausted in from six to twelve months.

The **prognosis** is invariably fatal.

The **treatment** is surgical.

CHRONIC PANCREATITIS.

Etiology.—This condition may be due to congenital syphilis, but, with this exception, little is known as to the etiology of the disease.

Pathology.—The pancreas may be enlarged or diminished in size. There is a great increase in the interstitial connective tissue, and the glandular structure may become changed or atrophied.

Symptoms.—There are no characteristic symptoms. There are commonly present digestive disturbances resembling those of chronic gastro-enteritis. The stools may be fatty. Jaundice is present in some cases, and is due to the pressure of the enlarged pancreas upon the common duct. There may be considerable epigastric pain and tenderness, and in some cases a sense of resistance can be appreciated over the pancreas. Emaciation and debility become progressive. In some cases glycosuria has been observed, and the disease may run the course of a severe diabetes. Ascites may develop in advanced cases.

The disease is chronic, but, from the insidious character of the symptoms presented, the precise duration of the disease cannot be determined.

The **prognosis** is grave, but not hopeless.

The **treatment** is to be supporting. The general nutrition of the patient should be improved in every possible way, and pancreatin or minced pancreas may be given, as indicated in all cases of diminished pancreatic digestion.

PANCREATIC CYSTS.

Retention-cysts may arise from the occlusion of Wirsung's duct by biliary or pancreatic calculi. The latter consist mainly of carbonate of lime. The duct may be obliterated by pressure from without or by cicatricial contraction. In rare cases cystomata may occur and may merge into malignancy.

Pathology.—The cysts, which may be single or multiple, are filled with an alkaline grayish fluid which emulsifies fats, converts starch into glucose, and, more rarely, digests albumin. These characteristics, however, are not peculiar to pancreatic cysts, and the older the cyst is, the less likely is the fluid to present these reactions. Hemorrhages into the cyst are common, so that the fluid becomes bloody or of a brown or chocolate color.

The **symptoms** are chiefly those of an abdominal tumor, which is of globular form, resistant and smooth, and in some cases fluctuating. The tumor first presents itself in the epigastrium or in the left hypochondrium, but may extend to fill the entire abdominal cavity. There may be a deceptive pulsation which is transmitted from the aorta. Pain is inconstant. There may be symptoms of pressure on the portal vein and on the bile-ducts, and cases of fatal rupture have occurred.

The growth of the cyst may be apparent in three weeks, but usually the course is chronic and extends over months and years. A sudden enlargement is suggestive of hemorrhage within the cyst.

The **prognosis** is generally good.

The **treatment** is surgical.

CANCER OF THE PANCREAS.

Etiology.—The growth is more apt to occur in men than in women, and in those past thirty years of age.

Pathology.—Cancer of the pancreas may be primary or secondary. Scirrhus is the most frequent variety. The growth may be limited to the pancreas or may spread to the stomach, the intestines, the peritoneum, or the liver.

The **symptoms** are often obscure. Dyspeptic symptoms, loss of flesh and of strength, and cancerous cachexia are constant and progressive. When the head of the pancreas is involved (in one-third of all the cases) jaundice invariably results. Ascites results if the portal vein be pressed upon. The patient may at any time vomit blood, from ulceration of the duodenum. The stools may be fatty; usually they contain undigested food. Glycosuria may be observed. Epigastric pain and tenderness are almost invariably present. A tumor is apparent in half the cases, and is of an irregular, more or less rounded form. The tumor is immovable, and may transmit a pulsation from the underlying aorta, so that in doubtful cases the patient should be examined in the knee-chest position.

The **duration** of the disease is rarely over one year, and the result is invariably fatal.

The **treatment** is entirely symptomatic.

Of other varieties of new growths of the pancreas, *sarcoma*, *cystic epithelioma*, and *syphiloma* have been described, but they are all exceedingly rare.

V. DISEASES OF THE KIDNEYS.

CONGENITAL MALFORMATIONS OF THE KIDNEYS.

There may be atrophy or congenital absence of one kidney, the other organ being considerably enlarged. Both kidneys may be fused into one horseshoe-shaped kidney, or into an irregular mass which may be displaced downward into the pelvis or either iliac fossa, or may lie in the middle line of the abdomen. These malformations must be remembered in operating on the kidney. Cases are on record in which calculi have been impacted in the ureter of a horseshoe kidney, with complete anuria and death.

MOVABLE KIDNEY.

Synonyms.—Floating kidney; Palpable kidney; Ren mobilis; Nephroptosis.

The kidney is retained in place by its fatty capsule, by the peritoneum, and by the renal vessels. Under certain circumstances it becomes movable, pushing the peritoneum before it. In rare cases it may be invested with peritoneum, which is reflected at the hilum to form a mesonephron; to such a condition the term "floating kidney" is limited by some. The right kidney alone is affected in 76 per cent. of all cases, both kidneys in 13 per cent., and the left kidney alone in 11 per cent.

Etiology.—Congenital cases are rare. As an acquired disease movable kidney is more common in women than in men, in the proportion of 9 : 1. It is most common in multiparæ, in the laboring-classes, and after thirty-five years of age. It may be due to congenital laxity of the peritoneal attachments, to the laxity of the abdominal wall due to repeated pregnancies, to tight lacing, to severe bodily labor, or to absorption of the fatty capsule. The kidney may, moreover, be displaced downward by tumors. The term "enteroptosis" has been applied to cases of prolapse of the abdominal organs, especially of the transverse colon, with movable kidney, and frequently with dilatation of the stomach.

Symptoms.—In the vast majority of cases there are no symptoms referable to the trouble, except that, should the tumor be accidentally discovered by the patient, nervous symptoms due to alarm and apprehension may result. In other cases there appear neurasthenic symptoms even to the verge of hysteria, with nervous indigestion, and in many instances accompanied by pain. This pain may be referred to the back, on the side of the movable kidney, and may radiate around the ribs, downward to the thigh, or forward to the epigastrium. The pain is aggravated or induced by exertion, jolting, or the erect posture. In some instances there may be merely a dragging feeling or a sense of weight or pressure. The pain may be the only symptom.

In "floating kidney" there may be, in rare instances, attacks characterized by intense abdominal pain, chills, vomiting, and symptoms of fever and collapse. The urine is usually diminished, high-colored, and contains an excess of uric acid or of oxalate of lime. These "incarceration-symptoms" are thought to be due to kinks or twists in the ureter or the renal vessels, but observations on this point are not definite. Care should be taken not to mistake these attacks for those of acute peritonitis, renal calculus, or appendicitis. Twisting of the ureter may give rise to temporary or permanent hydronephrosis, which may also be intermittent. Pyelitis may also result. A floating kidney may press on the bile-duct and cause jaundice, or on the vena cava and cause œdema of the legs.

Physical Examination.—The patient being placed in the dorsal position with the knees drawn up and the abdominal wall relaxed, one hand is placed behind in the lumbar region while the other hand is placed in the hypochondrium under the margins of the ribs. Bimanual palpation reveals a solid smooth tumor, of the size and shape of the kidney, which is freely movable. It is not tender except on firm pressure. The detection of a movable kidney of the right side may be aided by asking the patient to take a long breath, so as to depress the liver and thus push the kidney down into reach. There is often an advantage in getting the patient to assume the knee-chest position. At times a number of examinations are requisite before a positive diagnosis can be made.

Treatment is indicated only when the displacement of the kidney gives rise to symptoms. The simplest form of treatment is to put the patient in bed for a month and then let him go about with a binder and a pad to keep the kidney in place. This plan will work well in some cases, but not in others. In severe cases not relieved by this procedure surgical interference may be necessary, the kidney either being removed or being stitched in its proper place by sutures.

ANOMALIES OF THE URINARY SECRETION.

Albuminuria.

Both serum-albumin and globulin can escape through the capillaries of the Malpighian tuft and appear in the urine. In practice it is not necessary to discriminate between these forms, and they are both commonly described by the term "albumin." Albumin in the urine was formerly considered as a positive proof of organic disease of the kidney, but it is now known that not only may advanced nephritis exist without albumin, but that albumin may appear in cases without kidney-lesion, although in these cases it is supposed that there must be some failure in the nutrition of the epithelium of the capillaries of the tuft, allowing of the escape of albumin through them. Three distinct forms of albuminuria are recognized:

1. *Spurious Albuminuria.*—Urine admixed with blood (as in hæmaturia or in hæmoglobinuria) or with pus will give the reaction for albumin in proportion to the amount of blood or of pus present. Tube-casts do not appear.

2. *Functional or Physiological Albuminuria.*—(*a*) Albuminuria may follow severe muscular exertion, sustained mental effort, excessive albuminous food, violent emotions, or cold bathing. A few hyaline casts may be present, especially after exertion.

(*b*) *Hæmic Causes.*—Albuminuria may accompany any of the severe anæmias, purpura, scurvy, or syphilis. Under this heading may be included certain cases of transient albuminuria occurring during pregnancy.

(*c*) *Abnormal Blood-ingredients*, such as alcohol, bile-pigment, sugar, chronic lead- or mercury-poisoning, may induce the disease; it may also occur after the administration of ether or chloroform. Hyaline casts also may be present in bile-stained urine.

(*d*) *Neurotic causes*, probably from changes in the blood-supply, may give rise to the condition, as after epilepsy, apoplexy, tetanus, or injuries to the head, and with exophthalmic goitre.

(*e*) *Febrile albuminuria*, with pyrexia from any cause, persisting during the period of fever. Albumin is present in small quantities, and is due to slight changes in the glomeruli caused by the febrile process.

(*f*) *Cyclic or periodic albuminuria* is a form occurring in young adults, especially in boys, in which albumin appears at certain times of the day. It seldom, if ever, occurs after rest at night, but usually after exertion or after the principal meal of the day. The quantity of albumin is usually small, although it may be considerable, and transient glycosuria or occasional hyaline casts may be present.

In functional albuminuria there is neither high arterial tension nor hypertrophy of the left ventricle of the heart, unless from some intercurrent affection, and neither constitutional nor uræmic symptoms are present. Cases must always, however, be regarded with suspicion, especially if albumin be present in considerable quantities or if it be persistent. Albuminuria in persons over forty years of age usually indicates changes in the kidneys.

3. *Albuminuria with Gross Renal Lesions.*—(*a*) Congestion of the kidney, either acute or chronic.

(*b*) *Organic disease*, acute and chronic nephritis, amyloid disease, and tumors.

Tests.—The urine should be collected for twenty-four hours, and a specimen of this urine should be taken for examination. If the urine be turbid, it should be filtered, unless the turbidity be due to urates, in which case a little heat will clear the specimen.

Heat-and-nitric-acid Test.—The urine is boiled in a test-tube. If opacity result, it is due either to *albumin* or to *earthy phosphates.* On adding a few drops of nitric acid the opacity disappears if due to phosphates; if due to albumin, it is permanent. This is the best routine test and the most satisfactory.

Heller's Test.—Upon a small quantity of pure colorless nitric acid in a test-tube is allowed to trickle an equal amount of clear urine, so that it will overlie the acid. If albumin be present, a sharp white band will appear at the contact of the two liquids. A somewhat similar zone may be formed by the action of nitric acid on urates if in excess, so that the more insoluble acid urates are precipitated. This zone, however, is not sharply defined, diffuses itself into the urine above, and disappears on the application of heat. A haze due to mucin may also occur above the albumin zone, and may obscure the test.

Picric-acid Test.—A saturated solution of picric acid may be used as in Heller's test. Mucin, peptone, and certain alkaloids yield an opalescence with picric acid, but this opalescence disappears on heating.

Esbach's test is valuable in the quantitative analysis of albumin. The test-solution is made by dissolving 10 parts of picric acid and 20 parts of citric acid in 900 parts of boiling distilled water. After cooling, a sufficient quantity of distilled water is added to make a total of 1000 parts. The graduated tube is filled with urine to the mark *U*, and then with the reagent to the mark *R*. The liquids are mixed by slowly reversing the tube, and the coagulum of albumin is allowed to stand for twenty-four hours. The height of the sediment read on the etched scale indicates the weight of acid-albumin in grams per liter of urine.

Other tests for albumin are superfluous, and for them the reader is referred to books on urinary analysis.

HÆMATURIA.

Blood in the urine may come from the kidney, the pelvis of the kidney, the ureter, the bladder, or the urethra.

Renal hemorrhage occurs after injuries or falls; after acute congestion or inflammation; rarely from the atrophic form of chronic nephritis; from toxic agents, such as cantharides, carbolic acid, and turpentine; from embolism, thrombosis, or aneurysm of the renal vessels; from tubercular inflammation; from new growths; from calculous pyelitis; with malignant forms of acute infectious fevers, as hemorrhagic small-pox or "black measles;" with certain hemorrhagic diseases, as scurvy, purpura hæmorrhagica, or leukæmia; as evidence of vicarious menstruation; and in some young adults as a simple hemorrhage without known cause. It is also caused in the tropics by the parasites filaria sanguinis hominis and the Bilharzia. Malarial hæmaturia is endemic in certain of the Southern States.

Hemorrhage from the ureter usually implies the passage of a calculus.

Hemorrhage from the bladder is caused by injuries, rough catheterization, ulcers, ruptured veins, new growths, and calculi.

Hemorrhage from the urethra is caused by traumatism, foreign bodies, calculi, ulcers, chancroids, rough catheterization or injury, and gonorrhœa.

Diagnosis.—Blood in the urine imparts to it a red or brownish color and gives the reaction for albumin. Hæmaturia is to be distinguished from hæmoglobinuria by the presence of red blood-corpuscles. Microscopical examination usually renders other tests superfluous.

Heller's test for blood-pigment consists in boiling the urine with a solution of caustic potash until flocculi of phosphates fall; these flocculi assume a red color from the freed hæmatin.

The *guaiacum test* consists of the addition to the urine of a drop or two of tincture of guaiacum and two minims of ozonic ether. At the junction of the two fluids a blue line forms, which becomes diffused through the ether. Spectroscopic examination may reveal the single band of reduced hæmoglobin or the double band of oxyhæmoglobin.

Care should be taken to exclude the admixture of menstrual blood from the specimen obtained for examination, and bloody urine should not be confounded with the staining by rhubarb, logwood, and a few other dyes.

Determination of the Source of the Hemorrhage.—In blood from the kidney the blood and urine are intimately mixed, and there may be blood-casts, rendering the diagnosis positive. The color is often smoky. Blood from the pelvis and the ureter is frequently passed in clots which resemble leeches in form and color.

Blood from the bladder usually is passed with the last portion of urine. In washing out the bladder the water comes away blood-tinged, whereas if the source of hemorrhage were from the kidney the water would come away clear.

Hemorrhage from the urethra occurs in the first part of micturition, and blood frequently escapes in the intervals. Local symptoms aid in revealing the source of the hemorrhage.

Hæmoglobinuria.

Whenever, from any cause, the red blood-corpuscles are dissolved in the blood, the coloring matter thus set free is excreted as methæmoglobin in the urine, imparting to it a reddish-brown color which may in extreme cases resemble that of porter. The urine contains granular pigment and is albuminous, but usually no red blood-corpuscles are present. If present, their number bears no proportion to the intensity of the color of the urine.

Hæmoglobinuria is to be distinguished from hæmaturia by the absence of red blood-corpuscles; but care should be taken not to mistake hæmoglobinuria for bloody urine in which the corpuscles have been dissolved during ammoniacal decomposition. The urine reacts to Heller's test (see page 618) and gives the spectroscopic absorption-bands of methæmoglobin or, more rarely, of oxyhæmoglobin.

In all cases of hæmoglobinuria that have terminated fatally a secondary nephritis has been found.

Two clinical groups of hæmoglobinuria are described:

1. *Toxic Hæmoglobinuria.*—Dissolution of the red blood-

corpuscles can occur from the ingestion of certain drugs, such as potassium chlorate, carbolic acid, pyrogallic acid, naphthol, chloral, arseniuretted hydrogen, and muscarine. Hæmoglobinuria occurs after transfusion of blood, especially the blood of animals into the human subject, and after extensive superficial burns; it is said also to occur after exposure to cold. It may occur with certain infectious diseases, such as typhoid fever, scarlet fever, malarial fever, yellow fever, and syphilis, and an epidemic form of hæmoglobinuria in the new-born has been described, characterized by jaundice, cyanosis, and nervous symptoms.

2. *Paroxysmal Hæmoglobinuria.*—This form is characterized by the passage of blood-pigment in the urine in attacks. It is more common in men than in women, and it is usually seen in adults. Attacks may be induced by exposure to cold or as the result of bodily or mental exhaustion. Patients suffering from Raynaud's disease are peculiarly susceptible. Severe malarial poisoning may cause either hæmaturia or hæmoglobinuria, the changes in the urine frequently showing some regular periodicity. The attacks may be preceded by a chill and a rise in temperature, or the temperature may be subnormal. There may be yawning, headache, pain in the bones, vomiting, and cramp-like pains over the hepatic or lumbar region. The hæmoglobinuria rarely persists for more than one day; it then subsides, and is followed by slight jaundice in a considerable number of cases. Urticaria after the paroxysm is not uncommon. Ralfe describes cases in which paroxysms of hæmoglobinuria alternate with the same general symptoms, but with the passage of albumin and an increased amount of urea.

The **prognosis** of the toxic form of hæmoglobinuria depends upon the severity of the primary disease; otherwise the prognosis is good.

Treatment is unsatisfactory. Exposure to cold should be avoided; quinine should be given in malarial cases, and iodide of potassium is to be administered should a syphilitic history be obtained. During the paroxysm the patient should be confined to bed, kept warm, and given hot drinks.

PYURIA (PUS IN THE URINE).

Etiology.—1. *Pyelitis and Pyelonephritis.*—The pus is uniformly mingled with the urine, and the condition of the urine is unchanged after the bladder has been washed out. In calculous and tubercular pyelitis the urine is usually acid in reaction, but in pyelitis complicating cystitis the reaction is usually alkaline. Large abscesses of the kidney may suddenly discharge a large quantity of pus, and for days or weeks afterward the urine may be free.

2. *Cystitis.*—The urine is alkaline, often ammoniacal. The pus is passed with the last portions of the urine, and is mixed with thick, ropy mucus. The urine first obtained after the bladder has been washed out shows decided improvement.

3. *Urethritis.*—The pus is passed with the first portion of the urine, and may escape from the meatus in the intervals of micturition. Local symptoms of inflammation are usually evident.

4. *Leucorrhœa.*—The pus is small in quantity and is admixed with vaginal epithelium. This condition may be excluded by ordering a vaginal douche to be given before micturition, and by the use of the catheter.

5. *Rupture of an abscess into the urinary passages* is characterized by a sudden irruption of pus.

PEPTONURIA.

Peptone is never found in healthy urine. Traces of it are found in some acute diseases, in suppurative processes, and with disturbances of the digestion of albuminous substances, but the peptonuria possesses no diagnostic value.

Tests.—Peptone is not precipitated by heat or by nitric acid, but with picric acid there occurs a precipitate which is dissolved by heat. A supernatant layer of urine over Fehling's solution yields a rose-pink halo.

PHOSPHATURIA.

Phosphates occur in the urine as *alkaline* salts of sodium and potassium and as *earthy* salts of lime and magnesium. In urine undergoing ammoniacal fermentation the ammonio-magnesium salt or the triple phosphates may appear.

Phosphates are soluble only in neutral or acid urine, and are precipitated whenever the urine becomes alkaline. As they are less soluble in hot solutions, the phosphates are often precipitated by boiling, even in urine of a slightly acid reaction, and may be mistaken for albumin; but the specimen rapidly clears up upon the addition of acetic acid. If this acid be added to the specimen before boiling, precipitation does not occur.

Phosphates may appear in excess (up to 7 to 9 grams, whereas from 2 to 3 grams is the normal quantity) in those suffering from debility, dyspepsia, or wasting disease. There has been described a phosphatic diabetes characterized by polyuria, thirst, loss of flesh, and an absence of sugar in the urine. In some cases glycosuria or diabetes has followed this condition.

LITHÆMIA; URICÆMIA.

The daily amount of uric acid excreted depends largely upon the diet, varying from 10 to 30 grains, the relation of uric acid to urea being normally as 1 : 33. As to the production and antecedents of uric acid not much is known accurately, although it is supposed to be formed in the liver from ammonia and lactic acid. It is also unknown whether it represents a suboxidized grade of urea or whether it has an independent origin. Uric acid, being practically insoluble, is eliminated by the kidneys chiefly as the urates of sodium and ammonium, and to a less extent as the urates of potassium, calcium, and lithium. From these bases uric acid may be separated, forming the "red-pepper" or "brick-dust" deposits, which show characteristic appearances under the microscope. As conditions which cause the precipitation of uric-acid crystals from the urine Roberts mentions—(1) High acidity; (2) poverty in mineral salts; (3) low pigmentation; and (4) high percentage of uric acid. More commonly occurs the precipitation of amorphous urates, chiefly as the acid sodium urate, in the form of a pinkish deposit occurring as the urine cools. The urine is usually concentrated, of high specific gravity, and of excessively acid reaction.

The power which the blood possesses of holding uric acid in solution depends upon its degree of alkalinity. According to Haig, the excretion or the retention of uric acid can be regulated by increasing or diminishing the alkalinity of the blood. His theory is that agents increasing the alkalinity, finding a considerable quantity of uric acid in the liver, the spleen, and the tissues, render its solubility more perfect, so that it is taken into the blood and excreted by the kidneys. Pre-eminent among the drugs that increase the elimination of uric acid is sodium salicylate. Among those drugs causing retention, the most important are acids. Haig further believes that drugs affect only the excretion of uric acid, and have no influence whatever upon its formation.

The term "lithæmia" was first used by Murchison to designate symptoms due to functional disturbance of the liver and accompanied by an increased elimination of uric acid or urates; but it is impossible, in the present state of our knowledge, to state with any accuracy the pathology of the uric-acid diathesis. The diathesis bears a close relationship to gout, so that it has been termed "American gout." For its etiology and symptomatology see Irregular Gout.

OXALURIA.

Oxalate of lime is held in solution in the urine by the acid sodium phosphate. The crystals, which may be found deposited in small quantities under certain conditions, are easily recognized under the microscope. Oxaluria occurs after eating certain fruits and vegetables, as tomatoes, rhubarb, apples, pears, and cauliflower. It occurs also in gouty, hypochondriacal, and neurasthenic patients, as the result of imperfect oxidation-processes. Oxaluria is also said to result from acid fermentation of the urine within the urinary passages.

CHYLURIA.

Rare cases of chyluria result from some connection between the lymphatic vessels and the urinary passages, but the exact pathology is unknown. In the tropics chyluria is not infrequently associated with the presence of the filaria sanguinis hominis. The urine is milky in appearance and contains emulsified fat and serum-albumin. There may occur a spontaneous clot resembling blanc-mange, or the fat-globules may rise to the surface like cream. The microscope shows fine fat-globules which dissolve in ether.

INDICANURIA.

Indigo appears in the urine, not in the free state, but in combination as indoxyl-sulphate of potassium, which is a compound originally derived from indol. Indol itself is formed in the small intestine by the action of bacteria upon albumin. When concentrated acids are added to the urine containing the indoxyl-sulphate, indigo is liberated.

Indicanuria is frequent in all wasting and cachectic diseases associated with the excessive destruction of albuminoids. It may occur with tumors of the intestines and the pancreas, in intestinal obstruction, and in prolonged constipation. It is increased by a milk diet.

GLYCOSURIA.

It is a generally accepted belief that sugar does not occur in normal urine. The occurrence of glycosuria with pathological conditions will be considered elsewhere, under the same heading.

LIPURIA.

Fat may appear in the urine after an excessive quantity of fat has been taken with the food, with prolonged suppuration, with pancreatic tumors and degeneration, after phosphorus-poisoning, and in diabetic urine. The occurrence of chyluria has already been described. Lipuria may also occur with advanced Bright's disease and with pyonephrosis.

ACETONURIA.

Acetone, according to Von Jaksch, may occur with fevers, diabetes, cancer, inanition, in certain mental conditions, and as a form of auto-intoxication.

For the clinical and microscopical detection of the above-mentioned ingredients of the urine the reader is referred to books on urinary analysis.

ACUTE CONGESTION OF THE KIDNEYS.

Etiology.—Acute congestion of the kidneys may follow the taking of certain poisons (as cantharides), the removal of one kidney, or the sudden blocking of one ureter by a calculus, by over-exertion, or by surgical operations, especially on the bladder and the urethra.

Pathology.—The lesion consists in the temporary congestion of the blood-vessels of the kidney, allowing of the exudation of serum and the escape of red blood-cells.

Symptoms.—The urine, which is diminished in quantity or suppressed, according to the degree of congestion, may contain blood, albumin, and casts. Its specific gravity is not changed. The urinary symptoms may last for a few days with considerable prostration, and may then disappear; or the symptoms may continue, and the patient becomes more feeble and prostrated, passes into a typhoid state with delirium, and dies. These bad cases are those following the removal of one kidney, impaction of a calculus, or surgical operations on the bladder and the urethra.

The **prognosis** in mild cases is good. Repeated attacks induced by over-exertion or by irritant drugs may eventually lead to nephritis.

Treatment.—The patient is to be kept warm in bed, on a liquid diet, and the bowels are to be moved freely. Sweating should be induced by pilocarpine or by the hot pack or the hot-air bath.

CHRONIC CONGESTION OF THE KIDNEYS.

Etiology and Synonym.—Chronic congestion of the kidneys is induced by any cause preventing the free escape of blood from the renal veins, such as the pressure of abdominal growths or that occurring in the course of chronic congestion of the viscera due to heart disease or to emphysema. *Synonym:* Cyanotic induration.

Pathology.—The kidneys are normal or increased in size and are heavy and hard. The capsule is not adherent; the surface is smooth. The organ is congested, red, and livid, or the pyramids are red while the cortex is pink or white.

A considerable number of the glomeruli are large, their capillaries are dilated, and the cells covering the capillaries are swollen. Aside from a slight increase in the subcapsular connective tissue, the stroma is unchanged. Such kidneys are apt to develop chronic diffuse nephritis. In heart disease with visceral congestions chronic diffuse nephritis occurs in 60 per cent., chronic congestion in 40 per cent.

Symptoms.—The urine is diminished in quantity but is of good quality, the amount of urea to the ounce being rather increased than diminished. The specific gravity is normal or high. Albumin and casts may be present in small quantities, but are often absent. A continual precipitation of urates should excite suspicion of chronic congestion, and the heart and lungs should be carefully examined. The general symptoms are slight or unnoticed. Uræmic symptoms do not occur.

Treatment should be directed toward the disease causing the congestion.

ACUTE DEGENERATION OF THE KIDNEYS.

Etiology and Synonyms.—Acute degeneration of the kidneys is always secondary to the introduction of poisons into the body. It complicates poisoning by arsenic, mercury, and phosphorus, or by the organic poisons which result from severe infectious diseases or from injuries. Acute degeneration is usually found in the kidneys of those who have died from infectious diseases. *Synonyms:* Acute Bright's disease; Parenchymatous nephritis; Parenchymatous degeneration.

Pathology.—The kidneys are more or less enlarged; the capsules are not adherent; the surfaces are smooth; the cortex is usually thickened, and it may be either pale or congested. There are changes in the renal cells, especially marked in those of the convoluted tubes. These changes consist in (1) swelling, (2) granular infiltration with albuminoid matter and fat, (3) death of the cells, with desquamation, (4) a formation of hyaline masses in the cells, and (5) a growth of new cells to replace the dead epithelium. In severe cases there is added congestion of the blood-vessels, with exudation of serum. There are no changes in the stroma.

Symptoms.—The urine is diminished according to the severity of the disease. It may even be suppressed. Its specific gravity is unchanged. Albumin and casts are usually present, from the congestion and exudation, and blood-cells appear in severe cases. The general symptoms are usually obscured by those of the primary disease. In mild cases accompanying infectious diseases there are no symptoms, excepting the presence of albumin and casts in small amounts in the urine. In severe cases accompanying yellow fever and acute yellow atrophy of the liver, and following the ingestion of an inorganic poison, the urinary changes are marked: the patient becomes feeble, passes into the typhoid condition, and dies, apparently from the kidney-lesion. Dropsy is not noticeable in these cases.

The **prognosis** is good except in the severe cases. Albumin and casts may, however, persist for some time after the subsidence of the primary disease.

Treatment.—There is no treatment to prevent the degenerative changes. When exudation occurs the treatment of acute exudative nephritis is indicated.

CHRONIC DEGENERATION OF THE KIDNEYS.

Etiology and Synonyms.—The process is secondary to any of the mechanical causes of chronic congestion, to vicious modes of life, and to chronic alcoholism. *Synonyms:* Chronic Bright's disease; Chronic parenchymatous nephritis; Fatty kidney.

Pathology.—The kidneys are usually increased in size, although exceptionally they may be normal or even small. The surface is smooth; the pyramids are red; the cortex is white, yellow, or pink. There are swelling, granular degeneration, and fatty infiltration of the epithelium of the cortex. There are no changes in the stroma. The glomeruli are normal unless the degeneration be due to venous congestion.

Symptoms.—The quantity of urine varies in different patients and at different times; it may be increased, normal, scanty, or the urine may be suppressed. The specific gravity of the urine and the proportion of urea excreted are unchanged. Albumin and casts in moderate amounts are usually present. The patient becomes anæmic and loses flesh and strength. In bad cases he may pass into the typhoid state, with delirium and stupor. Dropsy does not develop. The disease may be followed by chronic nephritis.

The **prognosis** is not good, as the natural tendency of the disease is to progress.

Treatment is not satisfactory. Vicious and alcoholic habits are to be checked. The diet and the mode of life are to be regulated. The circulatory changes inducing venous congestion are to receive appropriate treatment.

ACUTE EXUDATIVE NEPHRITIS.

Etiology and Synonyms.—*Primary* cases may occur after exposure to wet and cold, or without assignable cause. *Secondary* cases accompany any of the severe infectious diseases or the puerperal state. *Synonyms:* Parenchymatous nephritis; Tubal nephritis; Desquamative nephritis; Catarrhal nephritis; Croupous nephritis; Glomerulo-nephritis.

Pathology.—The chief lesion is in the blood-vessels. From the tuft there is an exudation of plasma and of red and white blood-cells which infiltrate the stroma of the kidney and collect as casts and cellular masses in the tubes, from which they may be voided in the urine. The amount of exudation varies with the severity of the case. In severe cases there may be an over-production of pus-cells.

In mild cases the kidney shows no changes, the congestion having disappeared and the exuded material having been passed in the urine.

In severe cases the kidney is large, the surface is smooth, the cortex is thick and white or red and white, or the whole kidney is congested and succulent. Within the tubes, especially those of the cortex, are irregular masses of coagulated material, forming casts, and frequently white and red blood-cells. There may be dilatation of the cortical tubes, and their epithelium may be swollen, degenerated, or detached. The cells covering the tuft are usually swollen and increased in number. In other cases there is an excessive emigration of white blood-cells, which infiltrate the stroma and appear as small whitish foci in the cortex. The amount of serum exuded from the tuft is not proportionately increased, so that it is possible in this form to have little or no albumin and casts in the urine.

Symptoms.—*In mild cases* the patient complains of general malaise, with slight headache, loss of appetite, and perhaps some aching in the back. The urine is slightly diminished in quantity, the specific gravity is about normal, and albumin is present in considerable quantity, with hyaline, granular, and epithelial casts, sometimes with red and white blood-cells. The constitutional symptoms last for from one to two weeks; albumin and casts persist for four or six weeks and then disappear.

In severe cases there is fever, with prostration, loss of appetite, nausea, a pulse of high tension, and exaggerated heart-action. Anæmia is rapidly developed. There may appear the symptoms of acute uræmia due to contracted arteries—stupor, headache, dyspnœa, restlessness, muscular twitchings, or general convulsions. Dropsy may be developed, usually appearing first in the face. The urine is diminished in quantity, and contains albumin, casts, and cells in proportion to the severity of the inflammation. Constitutional symptoms last about four weeks. Albumin and casts continue in the urine for weeks afterward.

The cases with abundant production of pus-cells occur in children and in adults as a primary disease or secondary to measles, scarlet fever, and diphtheria. The invasion is sudden, with fever and prostration. Restlessness, delirium, headache, and stupor appear early in the disease and continue throughout its course. Dropsy is slight or absent. The patient loses flesh and strength, passes into the typhoid condition, and is apt to die.

The urine is not much diminished in quantity; its specific gravity is normal. Albumin, casts, and red and white blood-cells are usually present in considerable quantity, but they may not appear until late in the disease. In other cases albumin and casts are scanty or are absent altogether.

The **prognosis** of mild cases is good. The development of uræmic symptoms in the severe cases causes anxiety, but a decided majority of the cases recover completely. The cases with abundant production of pus-cells are apt to terminate fatally. It is possible for the disease to be followed by chronic nephritis.

Treatment.—The patient should be put to bed on a milk diet. In mild cases the liberty of the house may be allowed. The bowels should be kept open by calomel or by sulphate of magnesium in small repeated doses, and the skin should be rendered active by hot baths, daily cleansing, and skin-friction. Applications of wet or dry cups or hot poultices to the lumbar region may be of service. The cerebral symptoms during the early part of the disease, being due to contraction of the arteries with labored heart-action, should be controlled by arterial dilators. Of these, the best are aconite, chloral hydrate, nitroglycerin, and opium, in small repeated doses before the cerebral symptoms are marked, and in larger doses, hypodermically and by the rectum, during an attack. As the nephritis subsides the milk diet is replaced by solid food, iron and oxygen are given for the anæmia, and, if possible, the patient is sent to a warm, dry inland place until convalescence is complete.

ACUTE DIFFUSE NEPHRITIS.

Etiology and Synonyms.—Primary cases of acute diffuse nephritis follow exposure to wet and cold. Secondary cases complicate scarlet fever, diphtheria, and pregnancy. The disease is more common in children and young adults. *Synonyms:* Acute productive nephritis; Acute Bright's disease; Croupous nephritis.

Pathology.—The kidneys are large; the capsule is not adherent unless in cases of old standing, in which event the surface may also be roughened; the cortex is thick, white, mottled yellow and red, or congested; the pyramids are red. The microscope shows the same lesions as in acute exudative nephritis, with two additional features: First, a growth of connective tissue in the cortex; second, a growth of the capsule-cells of the Malpighian tufts. These changes do not occur throughout the entire kidney, but in symmetrical wedges in the cortex, following the line of the arteries. These wedges may be small or large, few or numerous, regular or irregular. Each wedge has the same general characters:

First, one or more arteries which run toward the cortex, the walls of which are thickened.

Second, the Malpighian bodies connected with the affected artery show an increased growth of capsule-cells, causing compression of the tufts. There is also a growth of the cells covering the vessels and within them, as in exudative nephritis. The tuft never returns to a normal condition, but in time the vessels are obliterated and the glomeruli are transformed into little balls of fibrous tissue.

Third, a growth of connective tissue in the stroma parallel with the affected arteries. This connective tissue is at first composed largely of cells; later the tissue becomes denser. If the growth of connective tissue be abundant, the tubes within the wedge become atrophied.

Between the wedges are seen the lesions of exudative nephritis; later there is developed a diffused growth of connective tissue.

The disease is serious not only from the disturbed functions of the kidney induced, but also because of the permanent character of the lesion.

Symptoms.—The invasion may be sudden or gradual.

The *acute cases* begin with fever and prostration, pain in the back, and frequent, scanty micturition. There are symptoms of acute uræmia, a pulse of high tension with exaggerated heart-action, or hypertrophy of the left ventricle, loss of appetite, nausea or vomiting, stupor, headache, muscular twitchings, or even convulsions. The patient becomes rapidly anæmic, and dropsy appears, usually first in the face; the dropsy may become general. The urine is smoky or bloody in color, scanty or even suppressed, of a low specific gravity, and contains albumin and casts in considerable quantities, with renal epithelium, and sometimes with pus-cells. These acute cases resemble cases of acute exudative nephritis, but the specific gravity of the urine is lower and the patient is more apt to die. At the end of about four weeks, however, the patient may apparently recover, although albumin and casts still persist in the urine. In course of time symptoms of chronic nephritis appear.

The *subacute cases* are more frequent. The first symptoms are often referred to the stomach—loss of appetite, nausea, and vomiting. In other patients dropsy is the first symptom complained of. Anæmia, headache, sleeplessness, and dyspnœa usually appear early in the disease. The urine is diminished in quantity, is of a low specific gravity, and may or may not contain blood. Albumin and casts are present, the former usually in considerable quantity. The patient becomes gradually worse. Acute uræmia from contracted arteries is shown by high-tension pulse, exaggerated heart-action or hypertrophy, with or without some dilatation of the left ventricle, headache, restlessness, and muscular twitchings or convulsions. Chronic uræmia is shown by alternating delirium and stupor, with a feebly acting heart and low arterial tension. The attack may last for weeks or months, and from it the patient may die. Other patients apparently recover, but the urine still contains albumin and casts and is of low specific gravity. There are usually subsequent attacks, which must be regarded as acute exacerbations of an established chronic nephritis.

The **prognosis** is bad, the patient dying either in the acute attack or from the chronic nephritis that follows. In many cases life may be prolonged for a number of years.

Treatment.—In the acute cases the treatment is the same as that for exudative nephritis, except that more care should be employed during convalescence, and prolonged residence in a warm inland climate should be recommended.

In the subacute cases the nephritis is best treated by rest in bed on a milk diet. The milk diet should be continued for a few weeks, and should then gradually be replaced by solid food. Later, residence in a warm, dry climate for some months at least should be insisted upon. Anæmia is to be treated by iron, oxygen, and fresh air. The dropsy is best treated by rest in bed on a milk diet. If the dropsy be persistent, diuretics, cathartics, and the hot pack or the hot-air bath may be employed. The condition of the heart and the arteries should be watched continually. If the arteries be contracted, with a pulse of high tension and an exaggerated heart-action, arterial dilators should be used. Among the best of these are nitroglycerin, chloral hydrate, opium, and potassium iodide. If the heart be feeble, with low-tension pulse, and if symptoms of chronic uræmia appear, digitalis, strophanthus, caffeine, or strychnine is indicated. In all cases the patient should be watched during convalescence, and the general health should be improved in every possible way. The selection of a suitable warm climate is of the very first importance in these cases.

CHRONIC BRIGHT'S DISEASE.

Although two forms of chronic nephritis are described, one with and one without exudation from the blood-vessels, practically the same lesions are found in both. The only real difference is that in one form exudation from the vessels is added. In chronic nephritis with exudation albumin is nearly constantly present in the urine, although it may be absent for short periods. In chronic nephritis without exudation albumin is generally absent, although it may be present at times in considerable quantity. The presence or absence of albumin seems to vary the clinical symptoms of the disease to some extent.

Chronic Diffuse Nephritis with Exudation.

Etiology and Synonyms.—Primary cases occur in young and middle-aged adults, being somewhat more common in males. The disease may follow acute diffuse nephritis, whether from cold, scarlet fever, or pregnancy, and it occurs after chronic congestion and chronic degeneration of the kidney. It may complicate syphilis, endocarditis, chronic phthisis, and prolonged suppuration, especially of bones and joints, and it is usually associated with amyloid degeneration of the kidney. *Synonyms:* Chronic parenchymatous nephritis; Large white kidney; Small white kidney; Chronic desquamative nephritis; Chronic tubal nephritis.

Pathology.—The kidneys are usually large, with smooth or roughened surfaces; the cortex is pale. This condition is spoken of as the "large white kidney." In other cases the kidney is small, with a pale cortex, forming the "small white kidney." In rare cases the kidney may appear to the eye to be normal. Microscopically the following changes are described by Delafield: "There is a very extensive growth of connective tissue in the cortex; the renal epithelium is swollen, granular, degenerated, fatty, broken, or flattened; the tubes contain coagulated matter, cast matter, or blood; the cortex-tubes are atrophied in some places, dilated in others.

"The glomeruli are changed in several different ways:

"1. There is a growth of the capsule-cells in such numbers that they compress the tufts. The cells covering the capillaries are also increased in size and number. The capsule-cells may finally be changed into connective tissue, and the tufts become atrophied.

"2. The glomeruli are of large size; the cells covering the capillaries are increased in number, so that the outlines of the capillaries are lost, but yet the capillaries are not compressed nor are the glomeruli atrophied.

"3. There is a growth of the cells which cover the capillaries and of the cells within them. Of the cells which cover the capillaries, the cell-bodies become very large, the capillaries are compressed, and the glomeruli eventually become atrophied.

"4. The walls of the capillary vessels become the seat of waxy degeneration, while the cells which cover them are increased in size and number.

" 5. If the nephritis follows chronic congestion, the capillaries are dilated, and there is an increase in the size and number of the cells which cover the capillaries.

" The arteries remain unchanged, or they are the seat of obliterating endarteritis, or there is a symmetrical thickening of all the coats of the artery, or all the coats of the artery are thickened and converted into a uniform mass of connective tissue, or there is waxy degeneration of the walls of the artery."

Symptoms.—1. *Changes in the Urine.*—The quantity varies at different times. When the inflammation is quiescent the quantity may be normal; during an acute exacerbation the urine is diminished or suppressed. In some cases, when the patient is doing badly, even if there be dropsy, the urine is increased. The specific gravity and the amount of urea to the ounce slowly diminish. The gravity varies usually between 1001 and 1012. Low specific gravity indicates extensive connective-tissue growth in the cortex or waxy degeneration of the capillaries of the glomeruli. Albumin and casts are almost constant in considerable quantities; they are increased during acute exacerbations, and at other times, when the lesion is quiescent, they may diminish, and may even disappear for short periods.

2. *Dropsy* is a prominent symptom, and is rarely absent. It may occur early or late, or only in periods.

3. *Anæmia* is most marked, and may even resemble pernicious anæmia. There is a peculiar pallor of the skin which is quite characteristic.

4. *Acute uræmic* symptoms, with contracted arteries, causing convulsions, etc., are not as common as in the cases without exudation.

5. *Chronic uræmia*, with soft, feeble pulse, delirium, and stupor, is common, especially toward the close of the disease.

6. *Headache*, restlessness and sleeplessness, loss of appetite, nausea, and vomiting frequently occur.

7. *Neuro-retinitis* and *nephritic retinitis* are not as common as in the cases without exudation.

8. *Dyspnœa* is nearly constant. It may be due to hydrothorax, to œdema of the lungs, to failure of the heart, or to contraction of the arteries. In many cases spasmodic dyspnœa occurring at night or in the early morning, and aggravated by lying down, may be the first symptom noticed.

9. The *tension of the pulse* is usually, but not always, increased. There may be hypertrophy or dilatation of the left ventricle, myocarditis, or feeble heart.

Course of the Disease.—The constant symptoms are (1) albumin in the urine, (2) dropsy, and (3) anæmia.

1. In some cases the symptoms are continuous, the patient dying from chronic uræmia or dropsy in from one to two years.

2. The symptoms come in attacks. Between the attacks the patient feels well, although the urine contains albumin and is usually of low specific gravity. The interval between the attacks may be weeks, months, or even years.

3. Some patients live for years with only pallor of the skin and albumin in the urine, feeling well otherwise.

4. There may be an attack of spasmodic dyspnœa in a time of supposed good health. Years may intervene before symptoms are developed.

5. There may be a history of chronic endocarditis, congestion of the kidney, or acute diffuse nephritis before the regular symptoms of the disease appear.

6. A few cases apparently recover, especially in children.

The **prognosis** is bad, although life may be prolonged for years.

Treatment.—The amount of urine should be increased by drugs or by so regulating the diet of food and liquids that the patient passes sixty ounces of urine a day.

The dropsy is to be treated by rest in bed, diuretics, and cathartics. Arterial dilators or heart-stimulants are indicated to meet the respective errors in the circulatory system.

Anæmia requires iron, arsenic, and oxygen. Prolonged residence, especially during the winter months, in a warm, dry climate is of great importance.

Acute uræmia with contracted arteries requires arterial dilators, such as nitroglycerin, chloral hydrate, opium, and potassium iodide, or bloodletting.

In chronic uræmia, with delirium, stupor, and a feeble heart, we employ the hot pack, heart-stimulants, and cathartics, but the results are not usually satisfactory.

During the acute exacerbation the patient should be put to bed on a milk diet and treated as if he had acute nephritis. Between the exacerbations plenty of outdoor life in fresh warm air may be advised, but not to the point of fatigue.

CHRONIC DIFFUSE NEPHRITIS WITHOUT EXUDATION.

Etiology and Synonyms.—This disease usually occurs in those over twenty years of age. It may appear in adults in a primary form as a gradual degeneration of the kidney; it often runs in families having a tendency to degeneration of the arteries. It may be caused by chronic alcoholism, lead-poisoning, gout, excessive eating and drinking, lithæmia, and constitutional syphilis. These causes are also factors in causing emphysema, endocarditis, endarteritis, and cirrhosis of the liver, with which diseases the nephritis is often associated. This form of nephritis also follows chronic congestion of the kidney, hydronephrosis, and chronic pyelitis. *Synonyms:* Chronic interstitial nephritis; Cirrhosis of the kidney; Granular kidney; Atrophic form of chronic diffuse nephritis; Gouty kidney; Renal sclerosis; Arterio-sclerotic kidney.

Pathology.—The kidneys are diminished in size; the capsules are adherent; the surfaces are granular; the cortex is thinned and of a red or gray color. There may be small cysts on the surface. Rarely the kidney is normal or even increased in size. The following microscopical changes are described by Delafield: " There is a growth of new connective tissue in the cortex, and also in the pyramids, which becomes more and more marked as the disease goes on. In the cortex the new tissue follows the distribution of the normal subcapsular areas of connective tissue, is in the form of irregular masses, or is distributed diffusely between the tubes. In the pyramids the growth of new connective tissue is diffuse. The tubes, both in the cortex and in the pyramids, undergo marked changes. Those included in the masses of connective tissue are diminished in size; their epithelium is flattened; some contain cast matter, many are obliterated. The tubes between the masses of new connective tissue are more or less dilated; their epithelium is flattened, cuboidal, swollen, degenerated, or fatty. The dilatation of the tubes may reach such a point as to form cysts of some size which contain fluid or coagulated matter. These cysts follow the lines of groups of tubes or are situated near the capsules. Of the glomeruli, a certain number remain of normal size, but with the tuft-cells swollen or multiplied. Many others are found in all stages of atrophy until they are converted into little fibrous balls. The atrophy seems to depend partly on the growth of tuft-cells and intercapillary cells, partly on the thickening of the capsules, partly on the occlusion of the arteries. If the chronic nephritis follows chronic congestion, the glomeruli remain large, but with a marked growth of tuft-cells; or they become atrophied, but with the dilatation of the capillaries still evident. The capillaries of the glomeruli may be the seat of a waxy degeneration. The *arteries* exhibit the same changes as are found in chronic exudative nephritis."

Complicating Lesions.—Hypertrophy of the left ventricle of the heart is almost constant, and affords corroborative aid in diagnosis. The hypertrophy may be followed by dilatation, chronic degeneration, or myocarditis.

General arterio-sclerosis is frequently found associated with the nephritis. Chronic endocarditis may complicate the disease. In some cases the heart-lesion is primary, being followed by chronic congestion of the kidney and by nephritis. In other cases the cardiac and renal changes occur together in the same patient and are due to the same causes, but are not directly dependent on each other.

Endarteritis with hypertrophy of the heart may give rise to cerebral hemorrhage.

Emphysema and cirrhosis of the liver may be found associated with the nephritis, all being types of chronic productive inflammation due to the same causes.

Pericarditis is not uncommon, and should be suspected in all nephritis cases dangerously sick with obscure symptoms. There seems to be an increased liability to gastritis and bronchitis.

Symptoms.—1. *Urinary.*—The urine is usually increased in quantity, is of a light-yellow color, has a specific gravity ranging between 1005 and 1010, and contains a diminished amount of urea to the ounce. Albumin and casts are usually absent, or albumin is present only in traces, especially in the early morning urine. During the acute exacerbations of the nephritis, or in the latter stages when the heart begins to fail, albumin and casts may be present in considerable quantities. In exceptional cases the specific gravity may be normal, or it may be exceedingly low, ranging between 1001 and 1003, with or without waxy degeneration of the vessels. During the acute exacerbations of the nephritis and during the attacks of contraction of the arteries the urine may be scanty or even suppressed. In rare cases there may be blood in the urine, or even profuse bleeding from the kidneys.

2. *Cerebral symptoms* appear in the majority of the cases, and are due to a variety of causes:

(*a*) Headache, usually frontal, and sleeplessness are common; the headache may be so severe that the patient is almost beside himself. There may be in various parts of the body neuralgic pains difficult of relief. Muscular twitchings and general convulsions are of serious import. There may be delirium, mild or furious, or stupor and coma. When these cerebral symptoms come in attacks the arteries are contracted, the heart's action is labored, and the temperature is raised. To this condition the name "acute uræmia" is given. From such an attack the patient may die, or he may recover, but the attack is liable to be repeated. These attacks may occur early or late in the disease, and such an attack may even be the first symptom noticed.

(*b*) Delirium and stupor may come on gradually in the latter part of the disease, with a feeble, low-tension pulse and a tendency to a subnormal temperature. These symptoms are due to chronic uræmia.

(*c*) There may be the symptoms of cerebral hemorrhage, coma, hemiplegia, and possibly of aphasia.

(*d*) There may be attacks of hemiplegia or aphasia, with coma which may persist until death or disappear in a few days. These symptoms appear to be due to endarteritis of the cerebral vessels, and not to any changes in the brain-tissue.

3. *Circulatory Symptoms.*—The pulse is hard and of an

increased tension. Thickening of the arterial wall from endarteritis may render the artery less compressible, and should not be mistaken for an actual increase in tension. A low-tension pulse is not a favorable symptom. The left ventricle is almost invariably found hypertrophied, and the second aortic sound is accentuated. Should dilatation or heart-weakness occur, the pulse will fall in tension and signs of venous congestions will appear, so that the condition will resemble that of a chronic heart-lesion. A great deal can be done by appropriate treatment to prevent dilatation from occurring.

4. *Respiratory Symptoms.*—Dyspnœa is frequently the first symptom noticed, appearing in spasmodic attacks aggravated by exertion or by the recumbent position. The attacks may last for minutes, hours, or days. The spasmodic dyspnœa appears to be due to an association of contracted arteries with a feeble or dilated heart. Dyspnœa more or less steady may also be due to chronic uræmia, anæmia, pleural effusion, or pericarditis. There may be Cheyne-Stokes breathing toward the close of the disease—a most unfavorable symptom. Bronchitis, especially during the winter, is exceedingly common; pleurisy and pneumonia are not uncommon. There may be sudden œdema of the glottis or of the lung, especially during acute uræmic attacks. Emphysema frequently coexists and adds its symptoms to the clinical history.

5. *Gastro-intestinal Symptoms.*—There may be attacks of catarrhal gastritis or of spasmodic vomiting, which may be severe and uncontrollable, endangering the life of the patient. Loss of appetite and dyspeptic symptoms are common. There may be severe diarrhœa, which to a certain extent appears to be compensatory, aiding in the elimination of the urea, and therefore it should not be checked too rapidly unless it be excessive and exhausting.

6. *Special Senses.*—Sudden blindness—"uræmic amaurosis"—may occur without retinal changes, and may be temporary or permanent. Neuro-retinitis or retinitis albuminurica, with white specks and flame-shaped hemorrhages, with or without thickening of the retinal vessels, may occur as the initial symptom, and many cases of nephritis are often first diagnosed by the ophthalmologist, to whom the patient applies for relief from the troubles in vision. Ringing in the ears with dizziness is not uncommon, and deafness may occur.

7. *Skin.*—There may be some puffiness of the ankles from time to time, but this symptom is not common, and dropsy does not occur unless it be the result of a failing heart. The skin is usually dry, and sweating is not common. In bad cases of uræmia urea may be excreted, giving a frosted appearance to the skin. There may be purpura in cachectic conditions.

8. *General Condition.*—The blood becomes anæmic, but this is not as marked as in the cases with exudation. The nutrition is affected, so that there is a gradual loss of flesh and of strength.

Course of the Disease.—At autopsy from some intercurrent disease the lesion may be found in a fairly advanced form without having given appreciable symptoms during the life of the patient.

2. Some patients go for years with a urine of low specific gravity, with a hypertrophied left ventricle, with accentuated second aortic sound, and with habitual increase of arterial tension, and yet feel perfectly well. Upon these symptoms, however, a diagnosis can with certainty be made. Any persistent high tension of the pulse with thickening of the arterial wall in a patient under fifty years of age points to cardio-vascular, and probably to renal, changes.

3. There may be, from time to time, attacks of acute

uræmia, with contracted arteries, increased blood-tension, and cerebral symptoms. The urine is scanty and contains albumin. Each succeeding attack is apt to be more severe, and between the attacks the general health depreciates and the patient becomes feeble and emaciated.

4. There may be no symptoms for a long time, the first evidence of disease being an attack of acute uræmia from which the patient may or may not recover, or of cerebral endarteritis or cerebral hemorrhage.

5. In some cases a gradual loss of flesh and of strength with digestive disturbances and low specific gravity of the urine may be the only symptoms until the time of the patient's death. The only symptom pointing to the kidney is the low specific gravity of the urine.

6. The kidney-lesion may give no direct symptoms; but should the hypertrophied heart begin to dilate and nervous congestion appear, the picture will be that of the last stages of heart disease, and the primary kidney-lesion will be overlooked. Many of the so-called cases of "idiopathic dilatation" arise in this way.

Prognosis.—The disease is absolutely incurable, but it is not incompatible with the enjoyment of a busy life for a number of years. Cases have been followed for ten or fifteen years. Much depends upon how much care the patient will take of himself, and how carefully dilatation and heart-failure are guarded against.

Treatment.—*General Treatment.*—Much good can be done by regulating the diet and the mode of life and by selecting a suitable climate. The diet should be light and nourishing, but not excessive. Meat should be taken but once a day, but the ingestion of fats should be encouraged. Alcohol should be prohibited; tea and coffee may be allowed in moderation. The urinary secretion should be kept free by drinking a certain amount of distilled water or some mineral water like Poland. The bowels should be kept freely open; the skin is to be kept active by daily baths and friction; and exercise in the open air should be encouraged in proportion to the strength. Much is gained by spending the winter months in some warm, equable climate. Severe mental or physical work should be avoided, so as to lessen the strain on the heart and the arteries. Anæmia is to be treated by iron.

Heart and Arteries.—A certain increase in tension is allowable. Excessive tension should be reduced by an occasional saline laxative and hot bath. Of the arterial dilators, the best are nitroglycerin (gr. $\frac{1}{100}$ three times a day, increased, if necessary, until the desired effect is produced), potassium iodide (gr. x three times a day), and chloral hydrate (gr. v–viij three times a day). Not only is the tension thus reduced, but headache, dizziness, and dyspnœa are often relieved in a most satisfactory manner. For restlessness morphia in small doses at night may be used. Should the heart's power fail and the tension be low, stimulants are needed as in cardiac disease—caffeine, digitalis, strychnine, and strophanthus. Opium is contraindicated in these cases, as small doses frequently cause fatal poisoning.

Uræmia.—Acute uræmia being due to contracted arteries, arterial dilators are indicated in free doses. The bowels should be opened rapidly, and in case of convulsion or coma bloodletting is frequently followed by brilliant results. Hypodermic injections of from $\frac{1}{8}$ to $\frac{1}{4}$ grain of morphia may be used in convulsions or coma with benefit, and for the convulsions whiffs of chloroform may be needed.

Chronic uræmia being due to retention of excrementitious substances, effort should be made to increase their elimination. The kidneys should be stimulated by digitalis combined with the saline diuretics; cups and poultices over the kidneys should be employed in bad cases. The bowels should be opened energetically, and sweating is to be induced by the hot pack or the hot-air bath.

Acute exacerbations should be treated on the lines laid down for acute nephritis.

WAXY DEGENERATION OF THE KIDNEY (AMYLOID KIDNEY).

Waxy degeneration of the kidney does not appear as a disease by itself, but as an added degeneration to the lesions of chronic diffuse nephritis, usually of the form with exudation. It occurs in cases with prolonged suppuration, especially of the bones, with syphilis and tuberculosis, occasionally with leukæmia, and in rare cases without apparent cause. It is associated with amyloid degeneration of the spleen and the liver.

Pathology.—There are usually the appearances of the "large white," more rarely of the "small white," kidney. The glomeruli are distinct and have a bacon-like lustre, staining mahogany-brown with weak, watery solutions of iodine, and red with dilute solutions of methyl-violet. The amyloid degeneration involves the vessels of the glomeruli, of the vasa recta, and occasionally of the membrane of the uriniferous tubules.

Symptoms.—The urine is usually abundant, pale, and of a low specific gravity. Albumin is usually present in considerable quantity, and there may be globulin. Tube-casts frequently include hyaline varieties which give the amyloid reaction. The general symptoms are those of the associated nephritis, together with the original suppurative or cachectic disease to which the amyloid changes are secondary. The diagnosis is aided by the detection of waxy changes in the liver and the spleen.

TUBERCULAR DISEASES OF THE KIDNEY.

Tuberculosis of the Kidney.

Miliary tubercles are frequently found in the kidney in cases of general miliary tuberculosis. The tubercles are small, are unaccompanied by inflammatory changes, and do not give rise to symptoms.

Tubercular Pyelonephritis.

Etiology.—The tubercle bacilli may infect the kidney through either the blood-vessels or the urinary passages. The infection may in rare instances be primary, but the ordinary cases are secondary to a tubercular focus elsewhere, especially in the bladder, the prostate, or the seminal vesicles. In many cases it is impossible to say whether a primary tuberculosis of the kidney has been followed by secondary tubercular lesions in the genito-urinary tract, or whether the kidney-lesions have followed infection creeping up the ureters from a primary focus below.

Tubercular disease of the kidney is twice as frequent in men as in women, and it is most frequent in those of middle age.

Pathology.—The lesion usually begins in the pelvis of the kidney. The pelvis becomes dilated and is filled with pus and cheesy material ; its walls are thickened, infiltrated by pus and tubercle-tissue, and its mucosa becomes increased in thickness. Tubercular infiltration extends to the kidney-tissue and rapidly undergoes cheesy degeneration and softening, so that the kidney becomes honeycombed with cavities. In advanced cases the kidney is converted to a cyst containing inspissated cheesy matter infiltrated with lime-salts. Both kidneys are usually involved, but the disease is usually more advanced on one side than on the other. In other cases one kidney alone is involved, the kidney of the opposite side developing the lesions of chronic diffuse nephritis with exudation usually with waxy changes.

In the majority of cases tubercular disease of the kidney is complicated by similar tubercular changes in the ureter and the bladder, and sometimes in the prostate gland and the seminal vesicles as well.

Symptoms.—1. *Urinary symptoms* consist in the frequent appearance in the urine of pus, cheesy material, fatty epithelial cells, and shreds of kidney-tissue. There may be occasional admixture of blood. These urinary changes also occur with calculous pyelo-nephritis, and so possess no absolutely diagnostic value. The discovery of tubercle bacilli, however, in the urine is an infallible sign of tuberculosis. The bacilli are best found in the small particles of cheesy matter present in the sediment. Albuminuria is usually more marked than can be accounted for by the pus. Tube-casts are of rare occurrence.

2. *Local symptoms* consist of pain and tenderness over the affected kidney. The pain may be dull and continuous or it may be paroxysmal, resembling renal colic; in the latter case it is due to the passage of lumps of cheesy material along the ureter.

3. An enlargement of the kidney may be appreciated in some instances, as in calculous or suppurative pyonephritis, but as the enlargement is rarely decided, it is with difficulty detected.

4. The *constitutional symptoms* are those of other tubercular diseases. Fever is rarely absent, and usually presents a remitting hectic character. Anæmia, emaciation, and weakness increase with the progress of the disease.

5. There may be complicating tubercular diseases elsewhere which add their characteristic symptoms. Acute miliary tuberculosis not infrequently develops.

Diagnosis.—The diagnosis from calculous pyelonephritis is made (1) by the presence of tubercular disease elsewhere, especially in the lower genito-urinary organs; (2) by the absence of a history of renal calculi; (3) by the presence of the tubercle bacilli in the urine.

Prognosis.—The course of the disease is progressive. The great majority of cases terminate fatally within two years, but it is possible for the disease to stop and the patient to recover.

Treatment.—Surgical treatment consists in the removal of the diseased kidney, and this should be done before other portions of the genito-urinary tract become infected; hence an early diagnosis is of the greatest importance, and it should be a rule to examine for tubercle bacilli in every case of persistent pyuria, so that incipient cases of tubercular kidney may thus be recognized.

SUPPURATIVE DISEASE OF THE KIDNEY (SURGICAL KIDNEY).

Etiology.—The germs of suppuration may gain access to the kidney—(1) through the abdominal wall, as with penetrating wounds; (2) by extension from neighboring abscesses; (3) through the blood-vessels, as in pyæmia and malignant endocarditis; and (4) through the ureter, as from cystitis or following operations upon the genito-urinary organs. The first two methods of infection are exceedingly rare. Infection through the ureter is favored by inflammatory conditions of the urinary passages, by pyelitis, whether simple, tubercular, or calculous, and by injuries and contusions of the kidney.

Suppuration of the kidney alone is termed "suppurative nephritis," but as the pelvis of the kidney is almost regularly involved, the name "pyelo-nephritis" is often applied. Should the pelvis of the kidney be distended with pus, the term "pyonephrosis" is not inappropriately applied.

The cases of suppurative pyelo-nephritis complicating tubercular disease and calculi in the pelvis of the kidney will be considered when discussing these diseases.

Pathology and Symptoms.—1. *Abscesses produced by emboli* may occur in the course of pyæmia and malignant endocarditis. Both kidneys are large, congested, and studded with small abscesses. The blocking of small terminal arteries by the emboli causes pyramidal areas of white infarctions which subsequently break down to form abscesses.

The symptoms of embolic abscesses in the kidney are obscured by those of the primary disease. Pus may not appear in the urine, as the abscesses seldom communicate with the urinary tubules.

2. *Idiopathic abscesses* occur without assignable cause, and it is unknown whether the suppuration begins first in the kidney or in its pelvis. One kidney becomes partially destroyed, and in the remaining portion abscesses are found. The pelvis of the kidney is inflamed and becomes distended with pus, constituting a pyonephrosis. The suppurative processes may extend to the perinephritic tissues.

The symptoms are at first obscure:

(*a*) Pus-symptoms are present, consisting of irregular fever, erratic chills, and cold sweatings. The symptoms of septicæmia or of the typhoid condition may become marked, or the patient will develop the waxy changes in the viscera that regularly accompany prolonged suppuration.

(*b*) Symptoms of a localized abscess consist of pain and tenderness over the kidney.

(*c*) Pus and broken-down kidney-tissue are present in the urine. The urine is frequently acid, the pus is uniformly mixed with the urine, and the character of the urine is unchanged after the bladder has been washed out. From time to time the pus escapes from the dilated pelvis in large quantities and appears in the urine.

(*d*) There may be a tumor, appreciable by palpation should the pelvis of the kidney be sufficiently distended by pus. The tumor may become reduced in size by the escape of pus in large quantities down the ureters.

3. *Suppurative Pyelonephritis from Cystitis.*—Both kidneys are swollen, congested, and studded with small foci of inflammation. The pelvis of the kidney is inflamed and coated with fibrin and pus. By the confluence of the suppurative foci large portions of the kidneys are converted to abscesses. The ureters may be normal or they may be inflamed. The lesions of an acute or chronic cystitis are almost always present.

In some cases there is an antecedent history of cystitis. At the time of the kidney-infection, the patient will develop septic symptoms—repeated chills and irregular fever with prostration. The urine will be diminished and will contain pus and blood from the bladder or kidney or from both. In some cases the urine is altogether suppressed. Prostration becomes more extreme, and the patient dies with pyæmic or septic symptoms.

4. According to Delafield, suppurative nephritis following cystitis due to enlarged prostate presents a different clinical picture. The patients are men, usually over fifty years of age, who have suffered from cystitis and an enlarged prostate gland. The first symptom is a diminished quantity of urine containing blood, or blood alone may seem to be passed. In other cases the urine is altogether suppressed. The patient becomes restless, anxious, and shows an increasing prostration; there are usually no chills, and there may be no fever. The patient may either die in collapse within a few days or may die from septic poisoning.

The **prognosis** is always fatal if both kidneys be involved. If one kidney alone be the seat of abscess, it may be removed and the patient may recover.

Treatment.—The preventive treatment is directed toward the cystitis and toward strict asepsis in all operations upon the urinary organs. When symptoms of suppurative nephritis occur, it is of the greatest importance to decide whether one or both kidneys be involved. The position of localized tenderness is to be considered, but the surest method of settling the question is by cystoscopic examination. If by the cystoscope purulent urine be seen to flow from both ureters, operative treatment is of no avail. If the pus be seen to flow from one ureter alone, the prospects of recovery after the operation are rendered more probable.

TUMORS OF THE KIDNEY.

Benign tumors consist of fibroma, lipoma, lymphadenoma, and adenoma. These benign growths are of little clinical interest.

Malignant tumors are sarcoma and carcinoma.

Sarcoma may occur in adult life, but it is more frequent in young children, and not infrequently it occurs as a congenital tumor. The ordinary form of sarcoma is the small-celled variety ; a rarer form, usually of congenital origin, is the rhabdo-myoma, which consists of sarcoma and striped muscular fibre. Sarcoma of the kidney grows rapidly, is very vascular, and frequently breaks down in places to form cysts containing blood and clots.

Carcinoma is usually of the encephaloid variety, and so frequently shows an alveolar structure that it has been described as “malignant adenoma.”

Secondary cancer of the kidney is not uncommon, with a primary growth situated in the testicle or the prostate gland. In the secondary form both kidneys are usually studded with small isolated nodules.

Primary malignant growths of the kidney frequently reach enormous dimensions, so as to cause symptoms by pressure upon the abdominal organs. Pressure on the ureter frequently results in a hydronephrosis. Secondary deposits may occur by extension or metastasis.

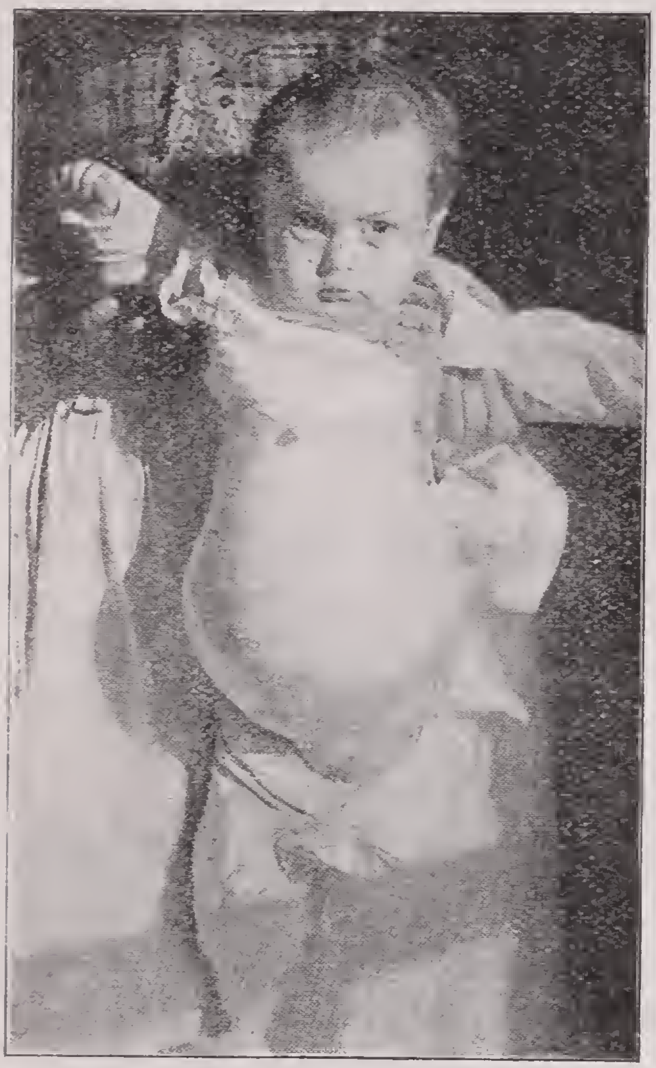


FIG. 49.—Sarcoma of kidney. Perfect health after operation (Abbe).

Symptoms consist of pain and tenderness, hæmaturia, cancerous cachexia, and the presence of an abdominal tumor.

Pain and tenderness are not constantly present. The pain may be steady and dull, radiating to the groin and the thigh, or there may be a paroxysmal pain, due to the passage of a blood-clot down the ureter, resembling the pain of renal colic.

Hæmaturia occurs in about half the cases, and may be the first symptom noticed. The blood, when present, is usually constant. There may be blood-casts of the pelvis of the kidney and of the ureter; these casts are very characteristic of cancer. The blood is usually passed in small quantities, although exhausting or even fatal hemorrhages may occur.

The symptoms of *malignant cachexia* become progressively marked. Anæmia is rendered more profound by the occurrence of hemorrhage; emaciation is extreme, and is in marked contrast to the enlarged abdomen.

The *tumor* is at first located in the region of the kidney, but it tends later to invade the whole abdominal cavity. It may be distinguished by the following peculiarities: (*a*) The surface often presents a series of convexities or knobs; (*b*) there is no respiratory movement of the growth, and (*c*) the hand may usually be thrust between it and the free border of the ribs; (*d*) the growth is not movable, as is a floating kidney; (*e*) percussion shows the colon passing in front of the growth; (*f*) a tympanitic zone is usually detected by percussion between the liver and the neoplasm; (*g*) rapid-growing tumors may yield a sense of fluctuation; (*h*) the renal tumor can usually be traced deeply into the loin.

In children the **diagnosis** of the growth from retroperitoneal sarcoma may be impossible.

The **duration** of malignant growths of the kidney is variable. The congenital cases may terminate fatally within a few weeks after birth. Usually in children the course is more rapid and malignant than in adults, seldom extending over one year. More rarely the disease may exist for a number of years before death occurs.

The **prognosis** is uniformly bad unless an early and successful nephrectomy be performed.

Treatment.—The results of nephrectomy for sarcoma in children are not good. The operation, however, should be performed if the diagnosis be made early in the disease, and in cases in which the disease is limited to the kidney. In adults the results of the operation are somewhat more encouraging. The risks of the operation itself are great, shock and hemorrhage being the imminent dangers encountered.

CYSTS OF THE KIDNEY.

Etiology and Pathology.—*Small retention-cysts* of the kidney are common in chronic nephritis; they may also occur in comparatively healthy kidneys.

Congenital Cystic Kidney.—The kidneys are enlarged and are converted into a mass of cysts of all sizes, separated by septa of connective tissue or of compressed kidney-structure. The cysts contain a clear fluid holding the urinary salts in solution, or the fluid may be brownish and turbid. Albumin, blood, uric-acid crystals, and cholesterin are often found in these cysts. Congenital cystic kidney is probably the result of defective development, but how the cysts actually originate is not known.

In adults multiple cysts may be found in the kidneys. The cysts contain clear or brownish serum or colored matter. Similar cysts are sometimes found in the liver and the spleen. The origin of these cysts is unknown, although it has been surmised that they are congenital.

Symptoms.—Cysts of the kidney give of themselves no characteristic symptoms except in rare cases of rupture. Symptoms of chronic diffuse nephritis are usually present, and death may finally result from uræmia.

Treatment is useless.

PYELITIS.

Etiology.—Inflammations of the pelvis associated with suppuration of the kidney, renal calculi, and tubercular disease have elsewhere been described.

Besides these forms, pyelitis may develop after the use of turpentine, cubebs, and cantharides, and from the irritation of saccharine or decomposing urine within the pelvis. Secondary pyelitis may complicate typhoid fever, diphtheria, and the exanthemata. Chronic catarrhal pyelitis may result from cystitis, from hydronephrosis, or from the continuance of an acute attack. In some cases the cystitis may develop without apparent cause.

Pathology.—The mucous membrane of the pelvis is thickened, infiltrated, and coated with mucus or with muco-pus. Phosphatic calculi may form as the result of the inflammation. In some cases the inflammation spreads to the kidney, so that a pyelo-nephritis results.

Symptoms.—Pain and tenderness are referred to the kidney. The urine is generally acid in reaction and contains pus, mucus, epithelial cells from the pelvis of the kidney, and occasionally blood. The pyuria may be constant or intermittent. If suppuration extend to the kidney itself, the symptoms of suppurative pyelo-nephritis or of pyonephrosis will be developed.

The **diagnosis** from tubercular pyelo-nephritis is made by the absence of tubercular foci in other parts of the body and by the absence of tubercle bacilli in the urine. The course of the tubercular form of pyelitis is more serious, and the disease usually terminates fatally. The diagnosis from calculous pyelitis cannot be made in all cases, but a long history of renal calculi affords an important clue to the diagnosis, and pyelitis is to be considered calculous if crystals of uric acid or of oxalate of lime be more or less continuously present in the urine.

Treatment consists in giving bland alkaline mineral waters or citrate of potassium in doses sufficient to render the urine neutral in reaction. The diet should be unirritating, and drugs that are capable of causing irritation or congestion of the kidney should not be given. If suppurative pyelo-nephritis occur, surgical treatment is indicated.

HYDRONEPHROSIS.

Etiology.—When any obstruction arises in the urinary tract, interfering with the free passage of urine, the pent-up urine causes an increasing dilatation of the urinary parts above the contraction. A contraction of the ureter may result from impacted renal calculi, from bends, from cicatricial contraction, or from external pressure, especially by new growths of the uterus or the ovaries. The lower orifice of the ureter may be stenosed in cancer of the bladder. In some cases congenital hydronephrosis results from congenital membranous obstruction or from abnormal valve-formations of the ureter. Double hydronephrosis, which results from strictures of the urethra, may also occur with enlargement of the prostate gland, or even with phimosis. Double hydronephrosis is regularly accompanied by dilatation of the bladder and of both ureters. The more gradually the obstruction is developed, the greater the degree of the dilatation; and it is important to remember that a sudden complete obstruction of a ureter, as by a renal calculus, does not lead to hydronephrosis, but to atrophy of the kidney.

Pathology.—The pelvis of the kidney is dilated; pressure-atrophy of the kidney-tissue results, and in the compressed kidney a chronic nephritis is set up. In advanced cases the kidney becomes converted to a cyst, in whose wall may be seen a thin rind of atrophied and compressed kidney-tissue. The fluid is thin and yellowish, and contains urea, uric acid, and sometimes albumin. It may be turbid from the admixture of pus. In cases of long duration the urinary salts may disappear, so that the fluid may not be characteristic. In extreme cases the sac may contain from ten to twenty quarts of fluid.

Symptoms.—The majority of cases give rise to no symptoms except the presence of a tumor, which first appears in the region of the kidney and enlarges toward the hypochondrium and the median line. Fluctuation may be appreciated in some cases. The colon lies in front of the cyst, and may be recognized by the tympanitic percussion-note obtained over it. The size of the tumor varies from time to time according to the amount of urine passed. In the rare cases of "intermittent" hydronephrosis the tumor may entirely disappear, with the discharge of its contents down the ureter. These cases, which are frequently congenital, appear to be due to a valve-like orifice of the ureter, which is opened only when the walls of the pelvis of the kidney are dilated and put upon the stretch. In other intermitting cases the ureter arises from the upper portion of the pelvis. Aspiration of the tumor differentiates hydronephrosis from solid growths, and the character of the aspirated fluid may confirm the diagnosis. In other cases intermittent hydronephrosis occurs with movable kidney. The growth of the tumor may give rise to pressure-symptoms, nausea and vomiting, and to shooting pains extending down the thigh. Uræmic symptoms may occur with bilateral hydronephrosis and in cases in which nephritis occurs in the dilated kidney. Suppuration may result in the formation of a pyonephrosis.

Prognosis.—Death may result from the primary disease, from secondary hydronephrosis, or from uræmia. Recovery may follow if the cause of the obstruction be removed; it may also follow operative interference.

Treatment is surgical. When the sac reaches large size the fluid may be removed by aspiration, or the sac opened and drained. The kidney may be extirpated as a last resort.

NEPHROLITHIASIS; RENAL CALCULI.

Etiology.—The solid constituents of the urine may be deposited in the kidney itself in several forms, to which the name "infarcts" has been incorrectly applied.

1. Uric-acid infarcts occur as reddish streaks at the bases of the pyramids in new-born children after the fourteenth day. They are not found in stillborn children.

2. Infarcts of sodium urate, with occasionally ammonium urate, appear as whitish lines at the apices of the pyramids in gouty kidneys.

3. Lime infarcts, seen in the kidneys of old people, appear as white lines in the pyramids.

The term "calculi" should more properly be confined to the concretions formed within the pelvis of the kidney. These calculi are common to all ages and are more frequent in males than in females. The occurrence of calculi of uric acid or of oxalate of lime is favored by gouty conditions and by functional disturbance of the liver. Phosphatic calculi are more commonly associated with inflammations of the pelvis of the kidney. Diet seems to exert no direct influence upon the formation of renal concretions, but the use of hard drinking-water containing lime seems to predispose to their formation. The deposition of uric acid, according to Roberts, is favored by the following urinary conditions: (1) High acidity; (2) poverty of salines; (3) low pigmentation; (4) high percentage of uric acid.

Pathology.—The important varieties of calculi are as follows: (1) Uric acid alone or combined with concentric layers of the urates; (2) uric acid with alternating layers of lime oxalate; (3) lime oxalate alone ("mulberry calculus"); (4) phosphatic calculi of the magnesium salts, of the ammonio-magnesium salts, or of both; (5) urates alone, especially in children; (6) cystin, resulting in a soft concretion of bees-wax consistency. A central nucleus is occasionally found, consisting of mucus or of a small blood-clot.

The ordinary size of the calculi varies from small gritty particles—"renal sand"—up to the size of rice-grains. In the pelvis of the kidney there may be found larger calculi of an irregular branching shape—the so-called "dendritic calculi." Calculi forming perfect stony casts of the renal pelvis have been described. Uric-acid and oxalate-of-lime calculi are more apt to be formed in both kidneys than are those of phosphatic origin.

Symptoms are produced by (1) the passage of the calculus down the ureter; (2) its retention in the ureter; (3) its retention in the pelvis of the kidney.

1. *Passage of the Calculus through the Ureter.*—Small concretions may pass without symptoms, or may give rise to twinges of pain in the side, the pain running down to the bladder. Larger calculi, entering the ureter, are pushed downward in jerks by the pressure of urine behind them, and give rise to the symptoms of renal colic.

Symptoms of Renal Colic.—(*a*) *Pain* is severe, steady, located in the side or the back, with cutting or tearing exacerbations which radiate downward to the groin and testicle. Radiation upward does not occur. The testicle may be swollen and retracted. The pain appears suddenly, and disappears with equal abruptness when the calculus drops into the bladder. The pain is often so intense that the pulse becomes rapid, feeble, and irregular. Syncope and vomiting are commonly observed. Convulsions may occur in children. In some cases there are chilly feelings with a moderate fever during the attack.

(*b*) *Urinary Symptoms.*—There is a constant desire to micturate, and the act may be painful from an associated spasm of the neck of the bladder. The urine, which is usually scanty, may contain blood. It may be suppressed either from functional inhibition or from previous calculous disease with atrophy of the opposite kidney. In some cases the urine is copious and limpid. After the attack dull, aching pain, with some tenderness over the kidney, continues for several days.

2. *Impaction of the Calculus within the Ureter.*—The attack begins as renal colic; the cutting paroxysms of pain gradually cease, and the dull ache alone remains for a considerable time, and finally disappears.

(*a*) The calculus may finally be passed after a period of impaction. This fortunate occurrence is more common if several calculi are impacted within the ureter. The passage of the obstruction is accompanied by the symptoms of renal colic, usually with blood in the urine, and may be followed by the passage of an excessive quantity of urine, especially if the other kidney have previously been disabled by calculous disease. In one case of the writer's, in which atrophy of the left kidney had taken place by a previous impaction of a calculus in its ureter, impaction in the right ureter occurred, with anuria of twelve days' duration. The final passage of two small calculi was followed by the excretion of eighteen pints of urine containing over five ounces of urea, the diuresis not being due to the drainage of a hydronephrosis.

(*b*) If the calculus remain, completely obstructing the duct, urinary secretion of the kidney will cease as soon as the pressure of the pent-up urine equals the blood-pressure within the renal artery. The kidney undergoes atrophy, and is converted to a little cyst in whose walls may be seen a thin shell of kidney-tissue. The cyst contains from one-fourth to one-half an ounce of clear serum, or the contents may consist of inspissated pus containing calculi and lime-salts. The ureter above the impaction becomes converted to a fibrous cord. *Hydronephrosis never occurs if the obstruction be sudden and complete.*

The symptoms of such an atrophy of the kidney depend entirely upon the condition of its companion organ. If the remaining kidney be in good condition, it will be able to do the extra amount of work required of it, and no symptoms will be developed. If the other kidney, however, be destroyed in like manner by previous calculous disease, or, as has happened, if there be but one kidney, then anuria develops. The characteristic of this obstructive anuria is that it may exist for a number of days without marked uræmic symptoms. Finally, at the end of one or two weeks uræmic symptoms develop, or the patient may suddenly die.

In some cases pressure-necrosis of the ureter at the point of impaction allows of perforation and of peritoneal sepsis.

If the impaction be incomplete, so that gradual back-pressure is exerted upon the pelvis of the kidney, hydronephrosis may be developed. This, however, is a slow and gradual process.

3. *If the calculi remain in the pelvis of the kidney*, they tend to grow larger and to assume the form of the cavity in which they lie. There results from their presence either a pyelitis, mild, severe, or suppurative, or dilatation of the pelvis (pyonephrosis), or a suppuration of the kidney (pyelonephritis).

Mild forms of pyelitis result in *pain*, of a dull, aching character, usually increased by exertion. The pain is usually referred to the kidney or to the back, but it may be referred to the opposite kidney—a point which should be remembered in operating upon such cases. From time to time there occur attacks of renal colic with its characteristic pain.

Tenderness over the kidney is usually detected by bimanual palpation.

The urine, which is generally acid in reaction, contains mucus, epithelial cells from the pelvis of the kidney, blood, pus-cells, and crystals of uric acid or of oxalate of lime. Hæmaturia is not profuse, but is apt to be persistent, and is regularly increased by exercise, so that the blood may be passed in considerable quantities.

From time to time there occur acute exacerbations of the pyelitis. There may be an initial chill; the temperature rises to 102° or 104° F. and is accompanied by severe and distressing pain in the back. Profuse sweating follows the decline of the fever. During the attack the urine becomes smoky or bloody and contains a large number of epithelial cells. This "intermittent renal fever," which may be mistaken for malarial fever, is identical with the intermittent hepatic fever due to gall-stones.

Severe cases of pyelitis may merge into those of suppurative pyelo-nephritis and pyonephrosis. The symptoms of pyelitis become aggravated, pus appears in the urine, and chills, fever, and other septic symptoms develop. If the opposing kidney be previously destroyed by calculous disease, pyelo-nephritis may lead to fatal uræmia. Other cases die in a septicæmic condition.

Treatment should be directed toward the following conditions:

1. *The habitual passage of uric-acid or oxalate-of-lime crystals.* Usually there is found some digestive error. The bowels should be kept regular; the functions of the liver should be stimulated by cholagogues and occasional doses of calomel. The use of dilute hydrochloric acid (♏xv in a glass of water to be drunk during meals) is often of the greatest service, especially in cases of oxaluria. A reduction should be ordered in fats, sugars, alcohol, and meat, but fruits, vegetables, and milk may be given freely. The most important part of the treatment is to enforce regular and systematic exercise; unless this can be done, the effects of dietetic and medicinal treatment may not be apparent.

2. *Attacks of renal colic.* The indication is to relieve the pain and spasm. The patient should at once be immersed in a hot bath, or hot applications may be applied to the abdomen. Morphine is to be given in $\frac{1}{4}$-grain doses hypodermically, and is preferably combined with atropine. The same caution attends its use as in biliary colic. The pain having a tendency to cease suddenly, the tolerance for the drug will also cease, and toxic symptoms may appear if the

drug be given in too liberal doses. While the patient is passing under the effects of the morphine whiffs of chloroform may be necessary to mitigate the agony. After the attack is over the urine for some days should be filtered through gauze or through a fine sieve to find the stone, and by analysis its chemical composition may be determined.

3. *If the stone be impacted in the ureter*, treatment is given with a view to relax the spasm of the wall of the ureter, and to increase the quantity of urine, so as to push the stone along.

Spasm is relaxed by continuous hot applications and hot baths, during which the abdomen may be kneaded gently, and by small doses of morphine and atropine. Increased secretion of urine is accomplished by raising the blood-tension by appropriate heart-stimulants, the preferable drug for this purpose being digitalis.

If medicinal treatment be of no avail, and if anuria persist, showing permanent disability of the other kidney, the stone may be removed surgically.

If the stone remain in the pelvis of the kidney, various forms of solvent treatment have been recommended. Citrate of potash is to be given in doses sufficient to keep the urine neutral in reaction. The treatment must be discontinued as long as the urine is alkaline or ammoniacal. According to Roberts, three conditions are necessary for success in this treatment: the calculus must be of uric acid; it must be of small size; and the urine must not be alkaline or ammoniacal, as otherwise sodium biurate or phosphate is precipitated upon the calculus, rendering further solution impossible.

Instead of the citrate of potassium, alkaline mineral waters may be given for the same purpose, but, according to Haig, the lithium waters are useless. For the oxalate-of-lime calculi there is no solvent treatment. If the pain be so severe as to interfere with the patient's earning a living, or if suppurative pyelo-nephritis or pyonephrosis develop, the kidney may be cut down upon and the calculi removed or the kidney extirpated. Care, however, should be taken that the other remaining kidney is competent, and not atrophied from previous disease or congenitally absent.

PERINEPHRITIC ABSCESS.

Etiology.—Suppuration of the perinephritic tissues may result from traumatism or from extension of suppuration from the kidney, intestine (especially the vermiform appendix), liver, or spinal column. Burrowing downward of a perforating empyema has occurred.

Symptoms.—As the disease is, properly speaking, a surgical one, but a brief description of the symptoms will be given. Pain and tenderness are present in the lumbar region. The pain is somewhat relieved by keeping the body immobile and by flexing the thigh. In rare cases the pain may be altogether referred to the hip-joint or to the knee. In the lumbar region there may be detected a tender, indurated mass which in the latter stages may yield a sense of fluctuation. The abscess may appear externally, or internal rupture in any direction may occur.

The constitutional symptoms are those of an internal abscess—chills, fever, and the gradual development of sepsis.

Treatment consists in opening and draining the abscess.

VI. CONSTITUTIONAL DISEASES.

ACUTE ARTICULAR RHEUMATISM; RHEUMATIC FEVER.

Etiology.—Rheumatism may occur at any time of the year, but it is more common in the spring months. Heredity is traced in 25 per cent. of the cases. One attack predisposes to successive attacks, and relapses are common. No age is exempt. It is very common in children, and it may even be a disease of intra-uterine life. It occurs especially in those leading a life of exposure, and the exciting cause may be wet and cold or over-strain of a muscle or a joint. The disease is rare in the tropics.

Rheumatism at certain times assumes epidemic proportions, and when this is the case the clinical features are apt to vary.

The following theories of rheumatism have been advanced; no one of them has been satisfactorily proven:

1. The *nervous theory*, that rheumatism depends upon disturbances of the nerve-centres presiding over the nutrition of joints.

2. The *lactic-acid theory*, that rheumatism is due to the presence of lactic acid in the blood, due to some perverted tissue-change of muscle.

3. The *uric-acid theory* of Haig, that uric acid formed in the blood may be deposited in the joints by diminished alkalinity of the blood. It has been supposed that lactic and uric acid in combination might produce the lesions.

4. The *theory of microbic infection* is based upon the generalization of the lesions, the involvement of the fibro-serous membranes so commonly involved in other known bacterial diseases, the constitutional predisposition seen in many patients, and the occurrence of occasional epidemics of the disease. No germ has yet been isolated, although experiments seem to confirm the theory of bacterial infection.

Symptoms.—1. *General.*—The onset may begin acutely with a chill and fever, or subacutely by shooting pains in the joints, malaise, and moderate fever. In rarer cases the joint-symptoms are the first symptoms observed.

The fever is rarely intense, usually under 103° F., and runs no typical course. Formerly its duration was from two to four weeks, but owing to improved methods of treatment it now rarely lasts more than from two to five days. In children the fever is but moderate, and it may even be absent. It is important to watch the temperature throughout the disease. A rise in temperature usually means a fresh invasion of joints, or some complication, such as endocarditis or pericarditis, while a fall generally implies a subsidence of the disease and modifies the therapy. The occurrence of hyperpyrexia will be noted later.

The pulse is full and dicrotic.

The urine is diminished, is of increased acidity and high specific gravity, and contains urates and an increased quantity of uric acid. Febrile albuminuria may be observed.

Cerebral symptoms are not seen except in cerebral rheumatism with hyperpyrexia or in over-dosing of salicylic acid.

The blood in rheumatism becomes rapidly anæmic. In the majority of cases there occur drenching sweats of a peculiarly sour odor, and the skin may show sudaminal vesicles.

2. *Inflammation of Fibro-serous Membranes.*—The joints are almost regularly involved, giving a distinct clinical type to the disease. In children, however, joint-symptoms are regularly less marked than in adults, and may be absent altogether (the "abarticular" form). The lesion is a simple serous synovitis. The synovial membrane is hyperæmic; its cavity is filled with serum and flocculi of fibrin. There is no pus-formation. There are often similar lesions in the sheaths of adjacent tendons. There are pain, increased by motion, and tenderness of the affected joints. The extent and character of the swelling depend upon the amount of synovial effusion and the involvement of the adjacent tendon-sheaths. The skin over the joint is usually hot, reddened, and not infrequently is œdematous. These symptoms are less marked in children, who may only show some rigidity to passive motion of the joint, and a continued position of flexion which is especially marked in the knees, and appears to be due to inflammation of the sheaths of the hamstring tendons, the joints themselves escaping.

Characteristic of rheumatic synovitis are the great rapidity of its development and subsidence, the involvement of many joints by jumps (fresh articulations being involved while those first attacked are recovering), and the rarity of its attacking one joint alone. Monarticular rheumatism is so uncommon that a diagnosis of rheumatism should always be made with extreme caution. The larger joints are especially liable to be attacked, but the small joints of the hands and the feet may be involved. Any joint may be attacked, but the temporo-maxillary articulation is so rarely involved as to throw doubt on the diagnosis, should this joint be affected. Symmetry of involvement is rare, the disease differing in this respect from acute rheumatic arthritis. Pain and swelling often persist after the acute process has subsided, and there may be some stiffness from adhesions within the joint-cavity. An acute attack may be followed by any of the forms of subacute or chronic rheumatism. Recurrences of acute rheumatism are exceedingly common, especially in the rheumatism of young people.

Subacute rheumatism represents a milder form of rheumatism. The constitutional and local symptoms are less intense, but the duration of the disease is longer than in the acute form, and the condition tends to become chronic.

Complications of Rheumatism.—1. *Cardiac Affections.*—The endocardium and the pericardium may be involved in mild as well as in severe attacks of rheumatism, and may even be inflamed without any involvement of the joints, as in the abarticular rheumatism of children. The liability to heart-complications is most common in children; this liability diminishes with increasing age. The heart-membranes are not usually involved after the first week, if absolute rest and a restricted diet be enforced.

(*a*) *Pericarditis*, which complicates from 10 to 20 per cent. of the cases, may occur alone or with endocarditis. The inflammation may be fibrinous, fibrino-serous, or purulent, and it is often associated with hyperpyrexia and delirium. The rheumatic pericarditis of children often runs an obscure course. The child grows pale and emaciated, and dies of exhaustion or of heart-failure without the development of either dropsy or dyspnœa.

(*b*) *Endocarditis* is more commonly a rheumatic lesion than pericarditis, and it appears in a large percentage of both mild and severe cases. Endocarditis may even be the solitary manifestation of the abarticular rheumatism of children. The mitral valve is the one most frequently affected. Valvular disease may not lead to serious consequences, or slow changes may ensue, resulting in valvular thickening and retraction. In a few cases there occurs an added infection of micrococci, resulting in malignant endocarditis.

(*c*) *Myocarditis* is almost regularly secondary to endocardial or pericardial changes.

2. *Pulmonary Affections.*—(*a*) *Pleurisy* usually results by extension from a pericarditis, and is therefore left-sided and more intense in the portion of the pleura in most direct contact with the pericardium. The pleurisy is usually fibrinous, rarely fibrino-serous.

(*b*) *Rheumatic pneumonia* rarely occurs except in connection with pericarditis, and is left-sided. The pneumonia may present certain peculiarities: (1) Absence of the critical fall of temperature; (2) frequent absence of cough; (3) rarity of rusty sputum; (4) great rapidity with which the physical signs clear up.

3. *Hyperpyrexia* is probably due to the action of the rheumatic poison upon the heart-centres, and is of comparatively rare occurrence. It appears more commonly in hot weather; it is more frequent in men than in women, and especially in those with weak nervous systems. It is unknown in childhood, the greatest liability being between the twentieth and thirtieth years. It is almost unknown after the third attack of rheumatism. In one-half the cases pericarditis is present. In an equal number of cases the joint-symptoms subside as the hyperpyrexia develops. The hyperpyrexia may complicate mild or severe cases. The onset of high fever may be sudden, or it may be preceded by headache, delirium at night (not due to salicylic acid), restlessness, hyperæsthesia of the skin, or excessive micturition. The fever may rise gradually or suddenly to 106°, 108°, or 110° F., or there may be merely a high range of temperature for a number of days, without any acute exacerbation. Prostration, delirium, and coma usually appear, and may terminate fatally.

4. *Cerebral complications* complicate the hyperpyrexia, and occur only in about 2 per cent. of the cases. Cerebral

symptoms may suddenly develop, or they may be preceded by the same prodromal symptoms as in hyperpyrexia. These preliminary symptoms, if proven not to be due to salicylic-acid poisoning, should excite grave apprehensions.

According to Duckworth, cerebral rheumatism assumes one of three clinical types:

(*a*) There is delirium, mild and wandering or so violent as to call for restraint, followed by semi-coma, coma, and death.

(*b*) The patient passes suddenly into coma, which terminates fatally, at times within a few hours.

(*c*) There are well-marked spasmodic symptoms, followed by fatal coma.

In the great majority of the cases of cerebral rheumatism hyperpyrexia is present, and if the temperature be over 106° F., a fatal issue is almost certain unless prompt treatment be adopted.

The pathology of cerebral rheumatism is obscure. The brain may be normal, anæmic, or congested. In rare instances there are found evidences of an acute meningitis.

5. The relation of *chorea* to rheumatism will be considered under the former disease.

6. *Cutaneous Complications.*—There may be a fine miliary rash, or an eruption resembling that of scarlet fever. Purpura is not uncommon, and the various forms of urticaria and erythema may occur.

7. *Rheumatic Nodules.*—Subcutaneous nodules, varying from a barely appreciable size up to that of a bean, may appear, attached to the tendon-sheaths, to the deep fascia covering the bony prominences, and to the cranium. Those attached to the tendon-sheaths are freely movable. During the early stages of their development they may be a little tender, but when of longer duration they are absolutely

painless. They are most numerous on the fingers, the hands, the wrists, and about the elbow-joints, but they may appear upon the patellæ, the spines of the vertebræ, the skull, and the clavicles. They may appear during an attack of rheumatism or after its decline, and they are more common in the abarticular rheumatism of children. They may last for weeks or months, and they are regarded as a positive indication of rheumatism. They consist of connective tissue undergoing rapid proliferation.

8. *Pharyngitis and Tonsillitis.*—*Pharyngitis* is not uncommon in rheumatic subjects, and is characterized by the amount of pain being out of proportion to the apparent degree of inflammation. Rheumatic pharyngitis may be practically considered as an erythematous affection of the fauces, of a nature similar to cutaneous erythema.

Tonsillitis occurs in repeated attacks in rheumatic subjects, and may complicate an acute rheumatic attack.

Treatment.—Except in very subacute cases, the patient should be put to bed, no matter how mild the case, and not be allowed to get up until the acute symptoms have entirely subsided. The diet should be of milk and farinaceous food, nitrogenous food being absolutely contraindicated. Lemonade or the alkaline mineral waters may be given freely. The patient should not be exposed to draughts, and should sleep between light blankets.

Local treatment to the affected joints is often grateful. The joints may be encased in cotton and rendered immobile by padded splints. Hot applications are usually well borne, hot solutions of 1 per cent. of acetate of alumina or of lead and opium being highly efficacious. Counter-irritation by the thermo-cautery or by small blisters about the joint sometimes relieves the pain. The joints may be bathed with chloroform liniment, or ichthyol ointment (ʒj : ℥j) may be applied. Ice-bags are at times more grateful than hot applications, and are much used on the Continent of Europe.

Constitutional Treatment.—Salicylic acid (gr. v–x), sodium salicylate (gr. x–xx), salol (gr. v–x), salophen (gr. v–x), and oil of wintergreen (♏. x–xx in capsule or in milk) may be given in the indicated doses every two hours until slight deafness and buzzing noises in the ears are experienced, after which the dosage is to be reduced gradually. This salicylic-acid treatment seems to have no effect in reducing the duration of the disease nor in preventing cardiac complications, but it exerts a specific effect upon the fever and the pain, the temperature falling to normal and the articular pains disappearing within from two to five days. It is said that relapses are more common under this than under the alkaline treatment, but the so-called "relapses" are probably recrudescences of the disease, as the symptoms of rheumatism tend to recur should the treatment be discontinued too soon or should too small doses of the salicylic acid be given. Toxic symptoms—gastric disturbances, delirium, cardiac weakness, albuminuria, and a tendency toward hemorrhages—should not appear if the patient be conscientiously watched.

The *alkaline* treatment seems to lessen the liability to cardiac complications. The treatment consists in giving alkalies in dose sufficient to keep the urine alkaline. For this purpose sodium bicarbonate in ʒj–ij doses should be given every three hours, in water rendered effervescent by the addition of the juice of half a lemon. The urine becomes alkaline usually within twenty-four hours, after which time only enough alkali is given to keep the alkalinity of the urine constant. The addition of quinine sulphate (gr. iij–v) to each dose of the alkali seems to increase its therapeutic value. The alkaline treatment may be given alone, but it is preferably combined with the salicyl compounds. Trimethylamine and benzoic acid or sodium benzoate have been recommended for rheumatism, but their action is inferior to that of the salicylates.

For the relief of pain opium may be necessary. Phenacetine is of service, and antipyrine, in 10-grain doses every three or four hours, is useful in controlling the pains of afebrile rheumatism.

Hyperpyrexia is to be treated by the energetic use of the wet pack or the cold bath, and no time should be lost in reducing the temperature.

The treatment of the cardiac and pulmonary complications is considered under their respective sections.

During convalescence iron is indicated to relieve anæmia, and the alkaline treatment should be continued for some weeks after the cessation of acute symptoms. Over-use of the joints should be avoided. During convalescence nitrogenous food should be interdicted.

PSEUDO-RHEUMATISM (SECONDARY RHEUMATISM).

Under this heading are included a number of forms of articular disease secondary to a variety of infectious diseases, usually of septic origin. Among the most common of these diseases are the articular affections complicating mumps, dysentery, scarlet fever, typhoid fever, puerperal fever, pyæmia, purpura rheumatica, syphilis, and gonorrhœa. The characteristics of these pseudo-rheumatic affections will be considered in connection with the diseases from which they originate.

CHRONIC ARTICULAR RHEUMATISM.

Etiology.—The chronic form may follow acute rheumatism, or the disease may be chronic from the start. Persons past the middle age of life, and whose occupation exposes them to wet and cold, are more liable to be attacked. The disease is greatly influenced by weather, and exacerbations may recur every autumn and last throughout the winter.

Pathology.—The synovial membrane is congested, and, together with the capsule of the joint, is thickened. The thickening may also extend to the fibrous structures about the joint. Slight erosions of the articular cartilages may occur in protracted cases. There may be deformities from the contraction of fibrous bands formed about the diseased joints (*rheumatisme fibreux*), and in advanced cases ankylosis of the joint may occur. There may be atrophy of the muscles about the diseased joint, from disuse, from centric nervous causes, or from neuritis. Muscular contraction is not uncommon. Osteophytic deposits do not occur as in arthritis deformans. There are no cardiac complications as in the acute form.

Symptoms.—Pain and stiffness characterize the affected joints, and acute exacerbations may occur, especially after over-exertion of the joints or during cold, damp weather. The affected joints are tender to the touch and are usually somewhat swollen. Synovial crackling may be appreciated on movement, and may readily be distinguished from the bony grating common to anthritis deformans. As a rule, many joints are involved, especially those exposed to fatigue in the patient's ordinary occupation. In working-women the small joints of the hand are usually favorite seats of the disease. Ulnar deflection of the fingers is common, but the deflection is not permanent except in the fibrous or ankylotic form. In rare cases the disease assumes a monarticular form, involving the hip, the knee, or the shoulder. The course of the disease is afebrile, and the general health does not directly suffer from the disease.

The **prognosis** is good for life, but is bad for recovery from the rheumatism.

Treatment.—The clothing should be warm, and exposure to cold, damp weather should be avoided. Salicylic acid is useless. A number of drugs have been recommended for this disease—potassium iodide, colchicum, arsenic, alkalies, guaiac, preferably combined with potassium citrate and tincture of cinchona—but the efficacy of internal treatment can seldom be demonstrated. Good results have been claimed for ichthyol (gr. v three times a day in pill form). The best results are obtained by improving the patient's nutrition (tonics, nutritious food, and cod-liver oil), by applying counter-irritation to the joints (thermo-cautery, iodine, blisters), and by sending the patient for the winter months to a warm, equable climate. Hot alkaline waters are particularly useful, and sulphur-waters have also been recommended. The thermal springs may be advised, together with a change of climate. Alkaline baths have been recommended, but they are not to be advised in the case of those over sixty years of age with atheroma and myocardial degeneration.

MUSCULAR RHEUMATISM.

Etiology.—The exact nature of this affection is unknown. The condition, which commonly occurs after exposure to cold and after over-use of the muscles, is more frequent in those of a rheumatic or gouty habit. One attack renders the patient susceptible to recurrences.

The **pathology** is unknown. The disease may be a mild form of inflammation of the muscles (myositis), or the affection may be entirely neuralgic.

Symptoms.—There is pain, dull and aching or sharp and cramp-like, in the affected muscles, and the pain is regularly increased by motion. The pain is generally relieved by pressure, but there may be decided muscular tenderness. The affected muscles may be contracted, so that the attitude of the patient frequently gives evidence as to the seat of the disease. Special names are given to muscular rheumatism according to the location of the difficulty. *Lumbago* affects the muscles of the small of the back and their tendinous attachments. *Torticollis*, or "wry-neck," usually affects the sterno-cleido-mastoid muscle of one side, but both muscles may be affected, and the muscles of the back of the neck are often involved. *Pleurodynia* involves the intercostal muscles of one side, and occasionally the pectorals, the latissimus dorsi, and the serratus magnus.

The **duration** of muscular rheumatism is usually brief, but the disease may at times run a subacute or a chronic course.

Treatment.—Rest for the affected muscles is of the first importance. As the pain begins to wear away, the subsidence of the disease may be hastened by massage. In some cases massage will cut short a pronounced attack, and the effects of the manipulations may be increased by the use of stimulating liniments. Strapping of the side gives relief in pleurodynia. Phenacetine combined with salol or with salicylic acid frequently cuts short an attack, and should always be used as routine treatment. Steaming by covering the affected muscles with damp cloths and passing a hot iron over them is a domestic remedy of established value.

A hot bath may be recommended. Rapid cure frequently follows "firing" with the thermo-cautery. The use of the constant current is also beneficial. Osler recommends acupuncture for lumbago, sterilized bonnet-needles three or four inches long being thrust into the affected muscles and retained from five to ten minutes. In many cases instant relief follows this procedure.

GOUT (PODAGRA).

Etiology.—Gout may be either inherited or acquired. Inherited gout is very common among the better classes in

England, Germany, and France, but is rare among the Irish. An inherited tendency is seen in 75 per cent. of the cases. Gout may be acquired by habits of ease, indolence, and dissipation, by over-eating and over-indulgence in alcohol, by deficient bodily exercise, and by any cause exhausting the great nerve-centres. It may be acquired through poisoning by lead, as Garrod found that 30 per cent. of his hospital cases of gout could be traced to this origin. Gout cannot always be traced to high living, as cases of "poor man's gout" are not uncommon, the disease being induced by improper hygiene, poor food, and indulgence in malt liquors. In America the acute forms of gout are uncommon, but the irregular manifestations of the disease are not infrequent. The disease occurs more often in men than in women. The inherited form usually appears earlier in life than the acquired form, which does not generally appear until the fifth decade.

Three theories of gout have been advanced:

1. *The Uric-acid Theory* (Garrod).—From renal insufficiency the excretion of uric acid is diminished, and the symptoms of gout arise from its retention in the blood and tissues and from the efforts of nature to expel it. The deposit of crystallized urates in the joints gives rise to the classical inflammatory symptoms. Haig has thus modified the uric-acid theory: The deposit of urates, according to his modification, is not due to their excessive production nor to deficient elimination, but is produced by all conditions associated with diminished alkalescence of the blood.

2. *The "Nervous" or "Nervo-humoral" Theory* (Duckworth).—Two conditions must be complied with: (1) The patient must possess the arthritic or diathetic habit from which gout and rheumatism arise, and (2) there must exist some functional disturbance of the nerve-centres to account for the sudden explosion of the symptoms. This "gouty

neurosis" may be inherited, acquired, intensified, or repressed. The causative effect of depressing physical and mental circumstances in inducing gout seems to lend corroboration to the nervous theory.

3. Ebstein believes that a local necrosis occurs in certain tissues from local nutritive disturbance, and that in the necrotic areas the urates are deposited secondarily. The nutritive-tissue disturbance may be inherited or acquired.

Pathology.—The blood contains an excess of uric acid. This condition, however, also occurs in leukæmia, and is not therefore distinctive. The joints usually affected are the great toe, the small joints of the hands and the feet, and, least frequently, larger joints without any marked order of preference. During an acute attack the synovial membrane is congested, the ligaments are swollen, and there is an increase in the synovial fluid. The articular cartilages appear as if whitewashed, from an interstitial deposit of the sodium urate, with occasionally the addition of the calcium salt. In advanced cases the cartilage becomes roughened and eroded and infiltrated with urates, while deposits of urates are seen in the ligaments and in the neighboring fibrous structures. The synovial fluid is often transformed to a pasty mass of urates.

Secondary inflammatory lesions consist of bony marginal outgrowths, and in ulceration by which the gouty masses may be discharged through the skin. The joint becomes distorted and immobile, and complete ankylosis may result. Gouty concretions or "tophi" are frequently seen under the skin, in the eyelids and the ears, and in other parts of the body.

Associated lesions are of great importance.

1. The kidneys may show deposits of sodium urate as whitish lines in the apices of the pyramids. Chronic diffuse nephritis is so commonly present that the name "gouty

kidney" has frequently been applied to this form of nephritis.

2. The arteries are frequently the seat of chronic endarteritis. Atheroma and calcareous deposits upon the aortic valves are not infrequent.

3. Hypertrophy and dilatation of the heart result from the nephritis, from the endarteritis, and from the atheroma of the aortic valves.

4. Emphysema with chronic bronchitis is almost constantly present in the chronic forms of gout.

5. Chronic gastro-enteritis or chronic colitis is frequent in long-standing cases.

Symptoms.—Gout may be described as occurring in acute, chronic, and irregular forms.

ACUTE GOUT.—There may be premonitions of an attack. These premonitions consist of twinges of pain, cramps in the calves, irritability of temper, and dyspepsia. A preliminary asthmatic attack may also occur. The urine is usually over-acid and concentrated, and deposits urates. There may be a temporary albuminuria or glycosuria. The quantity of uric acid eliminated before and during the early period of the attack is regularly diminished. The attack itself generally appears suddenly, usually during the early morning hours, with a characteristic pain in the metatarsophalangeal joint of the great toe, usually of the left side. The pain is grinding, throbbing, and excruciating, and is entirely disproportionate to the evident inflammation. The joint becomes swollen, dusky-red, shiny, and tender. The veins of the foot become turgid. As the attack wears off the foot usually becomes œdematous, and desquamation of the skin over the joint is observed. An initial chill is not uncommon. Fever amounting to 102° or 103° F. is almost regularly observed.

Other joints may become involved, especially the joint of the great toe of the other foot, but this extension of the disease is not common. A practical rule is that all cases of suddenly induced severe monarthritis should be suspected to be of gouty origin. In rarer cases the knee-joint is the one primarily involved.

The attack terminates favorably within a week or ten days, unless the response to treatment be exceedingly prompt, and the patient is left with a weakened, tender joint for some little time. Usually, after an acute attack the general health is markedly improved.

The terms "suppressed" and "metastatic" gout are applied to sudden and severe internal symptoms coincident with the rapid disappearance of the outward inflammatory signs. The following varieties may be described:

1. *Cardiac gout* consists of sudden pain in the heart, syncope, and heart failure which may be fatal. If the patient recover, dyspnœa and palpitation appear.

2. *Cerebral Gout.*—There may be mental confusion, delirium, or mania. Apoplectiform seizures with coma may occur. Temporary insanity has been observed. In some of these cases, however, the cerebral symptoms have been uræmic.

3. *Gastro-intestinal Gout.*—Pain in the stomach, nausea, and vomiting are not infrequent. Profuse diarrhœa may occur, with death in collapse.

4. *Vesical gout* is characterized by frequent painful micturition and hæmaturia.

5. *Testicular gout* is accompanied by painful swelling of the testis.

CHRONIC GOUT.—As the acute attacks become more frequent the local processes fail to leave the joints. The joints show various deformities, depending on the bony outgrowths from the periphery of the articular cartilages, from visible deposit of urates, and from enlargement of and gouty deposits in the superjacent bursæ. Synovial distention is less common in gout than in rheumatism. Crackling sounds are heard when the joints are moved. Pain and weakness are experienced in the affected joints, which first are those of the feet, then those of the hands. Tophi appear about the joints and in the ears, and may be discharged through the skin. The skin becomes soft and satin-like. Symptoms of the associated lesions appear—high-tension pulse from the arterial changes, hypertrophy or dilatation of the left ventricle, abundant urine of low specific gravity, and uræmic symptoms from the nephritis. Emphysema, bronchitis, and chronic intestinal catarrh complicate the course of the disease in almost all cases.

IRREGULAR or ABARTICULAR GOUT is extremely common, occurring as an inherited and as an acquired form. Various symptoms appear in different members of gouty families and among those whose habits are such as to predispose to gout. The symptoms are so varied and assorted that only a brief mention can be made of the most important:

1. *Cutaneous Symptoms.*—Eczema is frequent, especially the dry, scaly variety. Pruritus ani and hot itching feet at night are commonly the source of much distress.

2. *Gastro-intestinal Symptoms.*—Dyspepsia is the rule. Flatulence, over-acidity, constipation, coated tongue, "biliousness," and the symptoms of functional disturbance of the liver are usually present.

3. *Urinary Symptoms.*—The urine is usually over-acid and deposits urates, uric acid, and lime oxalate. Temporary glycosuria and albuminuria are not uncommon, especially in patients of advanced years. The symptoms of chronic diffuse nephritis without exudation may appear. Renal calculi are not uncommon, and are usually of the uric-acid variety. Urethritis may develop without gonorrhœal infection.

4. *Cerebral symptoms* are not uncommonly distressing. They comprise mental hebetude, loss of memory, irritability of temper, headaches of such severity as to suggest organic disease, vertigo, and sleeplessness. The eyesight is commonly blurred, and the eyeballs may be hot and itching.

5. *Cardio-vascular Symptoms.*—Vaso-motor symptoms are common, and consist of "hot and cold flushes" and sudden sweatings. The pulse is one of high tension. Palpitation is a common symptom. The arterial changes may lead to the symptoms of hypertrophied or dilated heart, atheroma of the aorta or of the coronary vessels, aneurysm, or sudden death.

6. *Pulmonary symptoms* include chronic bronchitis, emphysema, and asthmatic attacks.

7. *Locomotor Organs.*—Shooting pains, stiffness, and subtendinal bursitis are common. Cramps in the calves of the legs, burning feelings in the feet at night, and tenderness in the heels on standing may appear. Gouty neuralgia is not infrequent, and involves the sciatic nerve with greatest frequency.

8. *Eye-affections.*—A number of conditions may occur, among which iritis and glaucoma are the most important. Conjunctivitis is not uncommon, and may be due to gouty tophi in the upper lid.

Prognosis.—Gout is seldom the actual cause of death except in the rare visceral forms; but the disease is rendered serious by reason of the nephritis and the arterial sclerosis which so frequently complicate the disease, and it is upon these latter conditions that the prognosis depends.

Treatment.—During an acute attack of gout the foot should be elevated and wrapped in cotton. Hot applications are often of service, hot whiskey and water being a favorite application. Menthol may be used in alcoholic solution. The following prescription is used by Duckworth:

℞.	Atropinæ,	gr. iij ;
	Morphinæ hydrochloratis,	gr. xv ;
	Acidi oleici,	℥j.—M.

Sig. Paint over painful joint with a camel's-hair brush.

Colchicum has a specific effect on acute gout, and the wine or the tincture should be given in doses of from 15 to 20 minims every four hours, preferably combined with potassium citrate. A preliminary mercurial purge is usually given with advantage. The administration of colchicum should be watched carefully, and gastric distress and purgation should be avoided. In cases where colchicum fails or is not well borne, potassium citrate or acetate in 20- to 30-grain doses may be given every two hours, combined with diluent drinks. The preparations of salicylic acid are often employed, but their action is inferior to that of colchicum. Phenacetine or chloral hydrate may be given for the relief of the pain, but morphine is to be used with extreme caution. The diet during the acute attack should consist largely of milk and light farinaceous foods, and alcohol should be withdrawn unless especially indicated as a stimulant.

The treatment of chronic and irregular gout is largely by diet and hygiene. Starchy and saccharine food should be avoided ; beer and wines should be prohibited absolutely. Lean meats, eggs, fish, green vegetables, and milk should constitute the principal portions of the diet. Sweet fruits, berries, melons, and bananas are to be omitted from the dietary, but oranges and lemons may be allowed. The food should be simple, wholesome, and indulged in with moderation. If stimulants are needed, whiskey with water is the least injurious form. The patient should drink freely of pure water or of any of the alkaline mineral waters.

Hygienic treatment consists of daily baths with friction of the skin and regular systematic exercise. Exercise is one of the most satisfactory means of treating irregular gout, but it should not be carried to the point of fatigue.

The medicinal treatment is symptomatic. Lithia-water is of great service. An artificial lithia-water can be made by dissolving a 3-grain tablet of effervescing lithium citrate in each glass of table-water that is taken throughout the day. Constipation is relieved by occasional mercurial purgatives and by the steady use of podophyllin and rhubarb. Small doses of colchicum (♏v–x of the wine) with 10 grains of potassium iodide are of service during the more active manifestations of the disease. Quinine, guaiac, and the benzoates are also recommended. Iron and arsenic should be given for anæmic conditions. In obstinate cases much good is derived from a course of treatment at medicinal springs, such as the White Sulphur Springs or those at Saratoga, Carlsbad, Kissingen, or Homburg. The effects of the treatment are largely due to the improved dietetic and hygienic conditions that attend a course of treatment at these places.

ARTHRITIS DEFORMANS.

Etiology and Synonyms.—This disease may be primary in its origin, or it may follow rheumatism, gout, or gonorrhœal arthritis; 85 per cent. of the cases occur in women, especially in those at the time of the menopause. The influence of heredity is often marked, especially among the female members of a gouty family. Rarely the disease may occur in children. The true nature of the disease is obscure, but it seems probable that there is a nervous origin. This neuropathic theory is based upon the disease frequently following depressing nervous or mental shocks, upon the extreme symmetry of the lesions, and upon the atrophic changes occurring in the nails, the skin, and the muscles. In many respects the lesions resemble the arthropathies of locomotor ataxia. *Synonyms:* Rheumatoid arthritis; Rheumatic gout.

Pathology.—The joint-cartilage becomes fibrillated, soft, and velvety, and is worn away in the centre, exposing thickened, polished, eburnated bone-surfaces. At the periphery of the cartilages a lipping or a heaping up is observed, from which bony outgrowths form—the osteophytes. Immobility and deformity result from the interlocking and mutual obstruction of these osteophytes, but bony ankylosis does not occur except in the spinal column. The synovial membrane and the fibrous capsule become greatly thickened. Synovial distention is rarely extreme. Ligaments which pass in or through the diseased joints may be absorbed. In some cases the articular ends of the bones may be increased in length and thickness, but in old people the bones may become atrophied and spongy. Atrophic changes occur about the affected joints; the muscles undergo atrophy, the nails become brittle, the skin assumes a glossy appearance, and neuritis can frequently be demonstrated.

Symptoms.—Three clinical types of the disease may be described:

1. *Heberden's Nodes.*—The lesions involve the phalangeal joints, and little nodules (Heberden's nodes) develop on the distal phalanges. These nodes are composed of osteophytic outgrowths, and may show small cystic swellings at their summits, due to hernia of the joint-capsule. The joints may be swollen and painful after indiscretions in diet or when they are accidentally struck, but usually the chief symptom is limitation of motion. The phalangeal joints are somewhat enlarged, and may yield a bony grating on passive motion. The affection is incurable, but does not tend to advance.

2. *The Polyarticular Form.*—This form may develop

acutely and may be mistaken for acute articular rheumatism. It may be distinguished from the latter disease, however, by the symmetrical involvement of the small peripheral joints, by bony outgrowths from the articular ends of the bones, and by the uselessness of salicylic acid. The acute form of onset is most frequent in young women who have recently borne children.

In other cases the onset is *subacute.*

The *chronic* form is the most frequent. Premonitory symptoms are at times observed—numbness and tingling of the skin over joints, rapid and high-tension pulse, and a persistent pain in the ball of the thumb. The small peripheral joints are usually the first involved, and the lesions tend to advance steadily toward the trunk. The temporo-maxillary articulation is often involved. A characteristic feature is the extreme symmetry seen in the distribution of the lesion, for not only are corresponding joints simultaneously involved, but the lesions progress in them with identical rapidity. The joint-symptoms consist of pain, swelling, and limitation of motion. Pain is variable: it may be paroxysmal or of a steady, gnawing character, increased by motion and by warmth in bed, or there may be neuralgic pain or the pain of muscular cramp. The enlargement of the joints is due to thickening of the capsule, to bony outgrowths, and in some cases to synovial distention. Bony crepitus can usually be excited by passive motion. In time the joints become completely locked and immobile. Deformities are caused not only by distortion by the osteophytes, but also by muscular atrophy and spasm. The tendon-reflexes are usually increased, and ankle-clonus may be present.

3. *The Monarticular Form.*—This form is most frequent in old people and in men. The hip, the knee, the shoulder, and the spinal column are the parts usually affected. In

many cases there is a history of previous joint-injury. In old people especially a slight injury is often sufficient to set up what may be regarded as a senile change. The lesions are essentially those previously described, but great atrophy of the ends of the affected bones is characteristic of the disease in old people.

When affecting the hip-joint of the aged, the disease has been termed "morbus coxæ senilis." Pain, limitation of motion, bony grating, and shortening are observed. The gluteal region is flattened from muscular atrophy.

When affecting the spinal column, the disease has been termed "spondylitis deformans." Bony ankylosis is common, so that immobility of the vertebral column in the involved section results.

The **prognosis** is bad for recovery, although life is usually not shortened by the disease. Death occurs from intercurrent disease, from chronic nephritis, or from tuberculosis. The disease may be arrested at any stage, or it may progress so that the patient is practically crippled.

Treatment.—Arthritis deformans is practically an incurable disease. Much good can be done, however, by building up the general health and by the steady administration of arsenic. A temporary improvement may follow prolonged treatment by hot mineral baths or by drinking the waters of thermal springs. Massage may be of service in preventing the muscular atrophy of disease.

DIABETES MELLITUS.

The term "diabetes" should be limited to those cases in which sugar accumulates in the blood and is excreted in the urine, accompanied by constitutional symptoms. The term "glycosuria" should be applied only to those cases in which sugar appears in the urine in small amounts without constitutional symptoms, or appears only as a temporary condition.

Etiology.—The disease may be induced by long-continued indulgence in saccharine food, especially if the patient be of sedentary habits. In many cases the influence of heredity is marked. The disease may follow lesions or injuries of the brain or of the spinal cord, and it has been produced artificially by puncture of the floor of the fourth ventricle just behind the pneumogastric nucleus (Bernard's diabetic centre). Lesions causing atrophy in extensive disease of the pancreas have been followed so frequently by diabetes that a special form of "pancreatic diabetes" has been described. Psychical disturbances, such as worry, care, and depressing emotions, have been followed by the disease, and in some cases diabetes has succeeded certain infectious diseases, such as syphilis, gout, and malaria. Disturbances of the liver have also been adduced as causative factors. The disease is not as common in America as on the Continent of Europe. Men are more frequently attacked than women, and the greatest liability occurs in adult life. Children under ten years of age are usually exempt. Hebrews seem to be predisposed to diabetes, and the disease is more common among the higher classes.

To explain the disease a number of theories have been advanced. Nervous lesions, diseases of the pancreas and the liver, insufficient alkalescence of the blood-plasma, and deficient oxidation-processes have each been the subject of separate hypotheses, but no one theory has been established definitely.

Pathology.—A number of different lesions have been found, no one of which seems to be either constant or essential. They may be thus classified:

1. The *blood* contains an excess of urea, fat, and sugar. The fat-particles may be visible in coagulated blood; the

sugar may be raised from the normal 0.15 per cent. to 0.40 per cent.; glycogen may be found within the leucocytes.

2. *Nervous System.*—There may be tumors or cysts involving the "diabetic centre" in the medulla or involving the cerebrum. Anæmia, œdema, or atrophy of the cortical convolutions may be found, or congestion and thickening of the meninges. Perivascular changes in the brain and the cord may occur. There may be found a tumor pressing on the vagus. Peripheral neuritis is not uncommon. The sympathetic ganglia may be enlarged.

3. *Cardio-vascular System.*—The heart may be fatty or enlarged. Hypertrophy is not uncommon, and dilatation may be the cause of death. Endarteritis is frequently observed. Endocarditis may result from the irritation of the endocardium by the sugary blood.

4. The *liver* may be fatty or may be the seat of cirrhotic change. A peculiar form of pigmentary degeneration is described.

5. The *pancreas* may be firm and atrophied, may be the seat of pigmentary cirrhosis, or may be the seat of cancer, of cysts, or of fat-necrosis.

6. The *kidneys* are usually fatty. Glycogen is often found in the epithelium of Henle's tubes. Chronic nephritis is common.

7. The *lungs* frequently show the lesions of bronchopneumonia or of lobar pneumonia. Gangrene may occur. The lungs of diabetics are frequently the seat of tubercular inflammation.

8. The *mucous membranes* are usually the seat of chronic catarrhal inflammation.

The **symptoms** of diabetes mellitus begin insidiously. The first symptom noticed may be an unnatural thirst, the passage of too much urine, or a loss of flesh. In some cases the disease is first appreciated by an accidental examination of the urine. In rare instances it sets in rapidly. When the disease is developed the symptoms are changes in the urine, thirst, hunger, progressive emaciation, and the symptoms due to the complications.

1. The *urine* is usually pale, of a sweetish odor and taste, and is of a specific gravity ranging between 1025 and 1045, although a lower specific gravity may occur if the quantity of urine be enormously increased. The acidity is high. The urine irritates the genitals, causing intense pruritus, which may be the first symptom for which the patient applies for relief. Urea and phosphates are passed in excess, and acetone may be detected. A special form of diabetes has been described, "phosphatic diabetes" (Ralfe), in which phosphates are greatly increased, although the sugar may not be present constantly. Transient albuminuria is observed in about one-third of the cases. The quantity of urine passed varies between two and fifteen quarts, from four to six quarts being the quantity in average cases. In rare instances polyuria is not observed. The percentage of sugar varies up to from 1 to 2 per cent. in mild cases, and to from 5 to 10 per cent. in the severer forms. The total daily excretion of sugar varies up to from ten to twenty ounces, but it may exceed one to two pounds in the twenty-four hours. Sugar may temporarily disappear during the course of severe intercurrent diseases. The best methods of detecting and estimating the sugar are by Fehling's test and the fermentation-test. For details of these tests the reader is referred to works on urinary analysis.

2. *Excessive hunger and thirst* are observed in almost all cases. Thirst may be a distressing symptom. As a rule, the food is well digested, but from the excessive amount taken dilatation of the stomach may occur.

3. *Emaciation* is most marked in young subjects, in

whom the disease seems to run a more malignant course. The tongue is usually dry and glazed, the gums swell and bleed, and aphthous stomatitis may occur. Saliva is scanty. The skin is dry and scaly, but in some cases drenching sweats may occur, the perspiration being charged with sugar and lactic acid. Intense general pruritus is often distressing, and this symptom is always highly suggestive of either diabetes or internal cancer. Constipation is the rule.

4. *Complicating symptoms* are exceedingly numerous.

(*a*) *Cutaneous Symptoms.*—Boils and carbuncles are common, and suppurative processes are liable to follow operations. Purpura is frequent. Gangrene may occur, and is more frequently observed in the feet. The nails may atrophy, and the hair may fall out. The occurrence of pruritus has been mentioned.

(*b*) *Genito-urinary Symptoms.*—Irritation and pruritus of the external genitals are common. In men balanitis occurs. Impotence may be an early symptom. Cystitis may occur. The symptoms of a chronic nephritis may be added to those of the diabetes.

(*c*) *Pulmonary complications* are not uncommon. Lobar pneumonia, broncho-pneumonia, and gangrene of the lung may occur. Tubercular disease is common, and runs a rapid but somewhat insidious course.

(*d*) *Cardio-vascular Symptoms.*—Symptoms of arterial sclerosis are commonly present, and the heart is hypertrophied. Dilatation may ensue, and may lead to a fatal issue. Sudden death from fatty heart is not uncommon.

(*e*) *Nervous symptoms* are important.

Diabetic coma occurs especially in young subjects, and is usually associated with rapid emaciation. Preceding the onset the urine may be diminished in quantity, although the excretion of sugar remains unchanged. Three clinical forms of diabetic coma are encountered.

(1) After severe bodily or mental exertion the patient develops prostration with rapid and feeble heart-action, passes into coma, and dies within a few hours.

(2) The patient suffers for a few days from weakness, constipation, dyspnœa, and abdominal pain. Then develop headache, restlessness, delirium, great dyspnœa amounting to "air-hunger," cyanosis, and rapid and feeble heart-action. The breath has a sweetish odor resembling that of rotten apples. Coma terminates the disease within a few days.

(3) Neither dyspnœa nor prostration appears, but the patient complains of sudden severe headache, feels as if intoxicated, becomes rapidly stupid and comatose, and dies in a few hours.

The exact nature of diabetic coma is unknown. It has been ascribed to toxæmia from acetone or from oxybutyric acid. In other cases it has seemed to be due to uræmia or to fat-embolism of the branches of the pulmonary artery within the lung.

Peripheral Neuritis.—Mild forms of neuritis give rise to neuralgic pains, numbness and tingling, and muscular weakness. Severe forms cause lightning pains in the legs, loss of tendon-reflex, paresis of the extensors of the feet, and a characteristic gait. To this grouping of symptoms the term "diabetic tabes" has been applied. In other cases paraplegia has been observed, and both hands and feet may be affected.

(*f*) *Mental symptoms* consist of melancholia, and in some cases general paresis has resulted.

(*g*) *Eye.*—Cataract is common, especially in young subjects, and develops with great rapidity. Retinitis, hemorrhages in the retina, optic atrophy, and sudden blindness have occurred. There may be paralysis of the muscles of accommodation.

Prognosis.—Complete recovery cannot be expected, although a large number of patients enjoy good health for a number of years, and the disease in them may be controlled by diet. A considerable number of the cases die of heart failure, diabetic coma, pulmonary affections, or nephritis. A smaller number die exhausted and emaciated from the diabetes alone. As a rule, the older the patient the slower the course of the disease. In cases under forty years of age the prognosis is exceedingly grave. The cause underlying the diabetes must always be taken into account in rendering the prognosis.

The **treatment** is by diet and by drugs.

Diet.—Sugars and starches should be excluded, as far as possible, from the food. Saccharin or glycerin may be substituted for sugar. All fruits and vegetables that abound in starch should be prohibited; among these are potatoes, rice and cereals, flour- and starch-compounds, beans, peas, turnips, grapes, plums, apricots, pears, apples, melons, figs, berries, beets, onions, and asparagus. Beer, cider and champagne, and sweet wines should also be prohibited. Among other food-substances to be avoided are liver, crabs, lobsters and oysters, thick gravies, and soups. Theoretically, bread should be prohibited, and gluten bread be given instead, but, as this is not readily taken by the patient, its use cannot in all cases be insisted upon.

Among the articles that may be taken are butcher's meat, game, poultry, fish, clams, eggs, bacon, butter, cream, cheese, nuts, spinach, tomatoes, cabbage, cauliflower, lettuce, cucumbers and pickles, gluten, bran, and almond-meal bread, clear soups, lemons, tea, coffee, and cocoa.

As a rule, the diet should be modified gradually, one prohibited article after another being cut off. Care should be taken that the patient's strength is not too far reduced by the restricted diet.

Drug Treatment.—The following forms of treatment have been recommended, and are given in the order of preference: Clemens' solution (a 1 per cent. solution of arsenic bromide) may be given in 3- to 5-drops doses after meals. Opium is a valuable drug, and in diabetes there exists a tolerance for its use. Codeia may be given in ½-grain doses three times daily, and may be increased to from 6 to 8 grains in the day; or morphine may be given (gr. ¼ doses t. i. d.), and increased until the daily dose is about 5 grains. The 1-grain opium pill may also be used. Bicarbonate and salicylate of sodium may be given in 1- to 2-dram doses in the day. Sulphide of calcium (gr. ¼–ij four times a day), iodoform (gr. ½–ij four times a day), and antipyrine (gr. x–xx three times a day) are all occasionally of service. The alkaline waters of Carlsbad and Vichy may be beneficial. Jambul has also been employed. For the diabetic coma, venesection and intravenous injections of a 3 per cent. solution of sodium bicarbonate have been recommended; but recovery from the condition is exceedingly uncommon.

GLYCOSURIA.

Sugar may be found in the urine temporarily under the following conditions:

1. With various forms of poisoning, such as that by curare, morphine, amyl nitrite, alcohol, chloroform, chloral hydrate, and carbon dioxide.
2. With certain infectious diseases, especially diphtheria, cholera, typhoid fever, and epidemic cerebro-spinal meningitis.
3. Gastro-intestinal disorders allowing of faulty digestion of sugar and starches, and with functional disturbances of the liver.
4. In gouty patients with chronic diffuse nephritis.
5. From nervous causes, as neuralgia, concussion, cerebral hemorrhage, etc.
6. During pregnancy.

DIABETES INSIPIDUS.

Etiology.—Diabetes insipidus is most common in young adults, especially in men; it not infrequently attacks children. Congenital cases may occur. The influence of heredity is frequently well marked. The disease has followed injuries and diseases of the brain and the spinal cord, infectious diseases, sun-stroke, sudden mental excitement, and the rapid drinking of large quantities of fluid.

Pathology.—The exact nature of the disease is unknown; it is supposed, however, to be a vaso-motor disturbance of the renal vessels, or, in congenital or hereditary cases, to be due to unnatural permeability of the blood-vessels of the Malpighian tufts. There are no essential post-mortem lesions.

Symptoms.—The disease begins, insidiously or suddenly, with the excretion of an increased quantity of urine, of low specific gravity. From five to ten pints daily is an average quantity, but from thirty to forty pints may represent the daily secretion. The specific gravity of the urine varies from 1001 to 1004. Albuminuria and glycosuria are rare, although muscle-sugar, or inosite, has at times been found. The excretion of solids, especially of urea and the phosphates, may be increased in some instances. Thirst is inordinate, depending upon the amount of urine passed, but the appetite is rarely excessive. A variety of hysterical and neurasthenic symptoms may appear, but grave constitutional symptoms are usually lacking.

Prognosis.—Spontaneous cure results in a few instances. In the majority of cases the disease is intractable to treatment, although it does not tend to shorten life.

The **diagnosis** should be made from hysterical polyuria, the polyuria of nephritis, and diuresis from drugs. Diabetes is to be excluded by the absence of glycosuria.

Treatment is not very satisfactory. The most reliable drug seems to be valerian in full doses. Ergot has been recommended, but large doses are usually required. Good results occasionally follow the use of antipyrine in 15-grain doses every four hours. The bromides and arsenic have also been used with benefit. The constant galvanic current may be employed, one pole being applied to the back of the neck, the other pole on the lumbar region. Codeine is said to be of benefit, but the danger of the habit should preclude its use. In severe cases benefit may follow an exclusive diet of meat and hot water.

SCURVY (SCORBUTUS).

Etiology.—Devastating endemics and epidemics of scurvy have occurred from the earliest times among armies, among sailors on long cruises, and among the inhabitants of besieged cities, but the disease is now comparatively rare. Sporadic cases, however, are not infrequent, and the disease attacks young children more frequently than is usually supposed.

Two theories as to scurvy are advanced: (1) That it is a dietetic disease. It is known to occur from bad and insufficient food, from lack of variety in food, and from lack of fresh vegetables. Garrod ascribes the disease to lack of potassium salts; while Ralfe believes that the disease is due to a diminished alkalescence of the blood, from diminution of vegetable salts, as citrates, oxalates, and lactates, in the food. The development of scurvy is regularly favored by poor hygiene, damp dwellings, lack of sunlight, depreciated general health, depressing mental states, and overexertion. The disease is equally distributed between the two sexes. (2) The infectious theory is that scurvy is due to an unknown micro-organism, and that the dietetic and hygienic causes are only factors predisposing to infection.

Pathology.—The kidneys, the heart, and the liver show parenchymatous degeneration. The spleen is enlarged and soft. Hemorrhages are found beneath the skin, beneath the serous and mucous membranes, and in internal organs. Submucous hemorrhages may lead to ulcerations.

Symptoms.—The onset of the disease is usually insidious, although acute cases have been described. The symptoms are (1) general and (2) hemorrhagic.

1. *General Symptoms.*—There are increasing weakness, pallor, and emaciation. Palpitation with feeble and irregular heart-action are common, and a hæmic murmur usually develops. Mental depression and lassitude become extreme. The temperature is rarely elevated, and it may be subnormal. Œdema of the ankles may be observed. The urine is usually of high color and of high specific gravity, and the phosphates may be increased, Albuminuria is not uncommon. The breath is foul. The bowels are constipated.

2. *Hemorrhagic Symptoms.*—The gums become spongy, tender, and bleed readily. These changes, however, may not be observed in infants and in old people. The teeth tend to loosen and to drop out. Ecchymoses and purpuric spots appear in the extremities and spread to the trunk; they may arise spontaneously or after slight traumatism. Epistaxis is common, but hemorrhages from the lungs, the stomach, the intestines, and the kidneys are less frequently observed. Subperiosteal hemorrhages may occur, leading to pain, swelling, and immobility in the affected member, and necrosis of bone may follow, or separation of the epiphyseal cartilage may result in young children. Paraplegia or convulsions may be due to cerebral or meningeal hemorrhage.

Scurvy in children requires separate mention. The frequent occurrence of infantile scorbutus in America has been brought to our notice by the able researches of Northrup of New York. The disease occurs in infants, usually between the ninth and fourteenth months, and is almost regularly due to an exclusive diet of proprietary food or of condensed milk. In rare instances it has followed the use of milk too largely diluted. There is no evidence that sterilized or Pasteurized milk can originate the disease.

Symptoms.—The child becomes anæmic, irritable, and cannot bear to be handled or touched. The symptoms of rickets may coexist. The changes in the gums are almost constant, although they may not be carried to an extreme degree. The most constant symptom of infantile scorbutus is a painful fusiform swelling of the lower extremities, usually of the thigh. The swelling is regularly due to subperiosteal hemorrhages. The skin over the swelling is usually tense and shiny, is not hot to the touch, and pitting on pressure does not occur. The tenderness is exquisite and is increased by motion. As the swelling subsides thickening of the shaft of the bone may be appreciated. Fracture of the bones and separation of the epiphyses may occur. Pseudo-paralysis is an important symptom; it results from the pain caused by a contraction of the muscles pulling upon their tender periosteal attachment. There may be purpura, ecchymosis, and hemorrhage from the various mucous membranes. Hæmaturia may be an early symptom of infantile scorbutus.

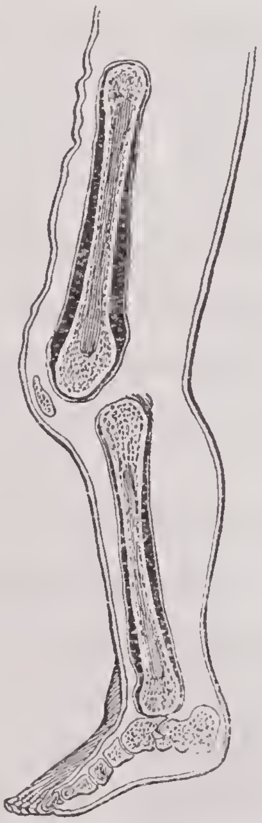


FIG. 50.—Vertical section of the thigh and leg in a case of infantile scorbutus. The dark areas along the femur and tibia represent subperiosteal hemorrhage (W. P. Northrup, from a specimen preserved in the museum of the College of Physicians and Surgeons).

The prognosis is good if proper diet and hygiene can be enforced. Death, however, may result from weakness, from heart failure, or from internal hemorrhages.

Treatment.—Sunlight, fresh air, a liberal diet of vegetables, and orange-juice are sufficient for a cure. Orange- or lemon-juice is almost a specific, and in children milk and orange-juice practically constitute the treatment. The gums may be pencilled with a strong solution of nitrate of silver, or mouth-washes of myrrh or astringents may be used. Anæmia is to be controlled by iron, fresh air, and proper diet. The hemorrhagic symptoms are to be treated on the principles laid down under the consideration of purpura.

RICKETS (RACHITIS).

Etiology.—Rickets is a disease of the first and second years of life, and is rare before the sixth month, although cases of congenital rickets have been described. The disease occurs especially in tenement-house children, from the combined effect of poor air, scanty sunlight, and defective food. The most common of the dietetic errors that may lead to the disease are premature weaning, the use of artificial and proprietary foods, especially those composed mainly of farinaceous and starchy ingredients, irregular feeding, prolonged lactation, and nursing during pregnancy. Children of weak or vicious parentage seem to be predisposed to the rachitic condition. The disease is more common among negroes and in European cities than among American children. The connection between syphilis and rickets has not been proven satisfactorily. Rickets is often delayed until the third or fourth year, and then may appear as a sequel to some infectious disease such as measles. The disease known as "acute rickets" is now supposed to be scorbutus.

Pathology.—The lesions are seen in their fullest development in the long bones and in the ribs. The epiphyseal cartilages undergo rapid proliferation, and form thick, soft cushions, which by their projection may present evident bulging. The periosteum strips readily, and subperiosteal tissue is seen to be soft and vascular, resembling spleen-pulp. This subperiosteal thickening is best marked in the middle of the shaft of the bones, giving to them a spindle shape. There is regularly delay or arrest of ossification-processes, and in the bones the organic ingredients may be reduced to two-thirds of their normal proportions. Rachitic bones are regularly soft, spongy, and vascular, especially near the epiphyses and beneath the periosteum. From the bone-softening various deformities arise—the box-like cranium, spinal curvature, deformed pelvis, knock-knee, bow-legs, and the like. In time there occur in the cartilages and in the subperiosteal tissues reconstructive changes resembling the callus-growth upon fractures, and the bones themselves become hard. In this way permanent deformities result. The liver and the spleen are usually enlarged in rickets.

Symptoms.—Certain *prodromal symptoms* may precede evident changes in the bones. Gastro-intestinal disorders are usually present, and a peculiar form of diarrhœa limited to the first part of the day, with scanty colorless stools, has been described. There may be attacks of screaming at night, and restlessness with an intermittent temperature and splenic enlargement. Profuse night-sweats about the neck and the back of the head are highly suggestive of incipient rickets. There is often such extreme soreness of the body that the child cries when handled. This diffused soreness is a most suggestive symptom. The child becomes flabby and anæmic, and usually emaciates. There may be "pseudo-paresis," from a combination of muscular weakness and disinclination to move on account of the general soreness and tenderness.

Nervous symptoms often are present. Attacks of spasm of the larynx are not uncommon. Convulsions, either general or partial, may appear at intervals; the convulsions may be fatal. Tetany and carpopedal spasms may be observed. The child is irritable and nervous.

Symptoms due to changes in the bones are seen first in the ankles, wrists, ribs, and cranium. The epiphyseal ends of the bones are swollen; the bones are liable to curvature, and "green-stick" fractures may be caused by slight injuries. Curvature of the spine (rachitic scoliosis and kyphosis) and pelvic deformities may ultimately result. The thorax shows noticeable changes. The swellings at the junction of the ribs with their cartilages produce visible nodules, to which the name "rachitic rosary" has been applied. The lateral portions of the thorax are frequently drawn inward at the portions corresponding with the insertion of the diaphragm. "Pigeon-breast" may occur. The changes in the thorax may be so marked as to interfere with proper chest-expansion, so that pulmonary affections trifling to the non-rachitic may become serious diseases in these children. The clavicles may be distorted or fractured.

Characteristic changes become apparent in the cranium. The fontanelles remain unclosed until the second or third year of life, and the occiput may be so thin and yielding that it can be pressed in like parchment. To this latter condition the name "craniotabes" has been applied. The head becomes large and square. The occiput is flattened, the biparietal diameter is increased, the frontal bones are broad and prominent. The square, box-like cranium may resemble hydrocephalus, but in rickets the child is mentally bright.

The maxillary bones are small and narrowed, and the vaulting of the roof of the mouth is increased. Dentition is late, and the teeth may be ill formed, small, or irregularly crowded. From the diminished growth of the bones the child may be stunted and dwarfed.

The **prognosis** is good for the disease itself, but many rachitic children fall victims to gastro-intestinal or pulmonary disorders. The prognosis should also regard the ultimate effect of the thoracic spinal and pelvic deformities.

Treatment.—The most important treatment is improvement of the general condition by proper diet, good air, sunshine, and salt-water baths. Anæmia is to be treated by syrup of the iodide of iron, and digestive disturbances should receive prompt attention. The child should lie on a hair mattress or pillow, and should not be allowed to walk so long as the bones are soft. Theoretically, preparations of lime, such as the calcium lactophosphate, are indicated, but as a matter of experience they do very little good. The drug *par excellence* is phosphorus, which should be given in doses of gr. $\frac{1}{150}$ three times a day in cod-liver oil. The deformities ultimately require special gymnastic exercises for their correction, or orthopædic treatment.

PURPURIC DISEASES.

Under this heading are included a number of diseases having one symptom in common—the extravasation of blood under the skin. Small hemorrhagic spots are known as "petechiæ;" larger hemorrhages are called "echymoses." Various degrees of intensity are met with in each variety of purpuric disease. The mildest form consists of subcutaneous hemorrhages alone; the severer forms include as well hemorrhages from free mucous surfaces and visceral hemorrhages. Thus purpura may be a trifling, a serious, or even a fatal disease. The following classification may be adopted, although it should be remembered that transitional forms are frequently encountered: 1. Symptomatic purpura; 2. Purpura rheumatica; 3. Purpura hæmorrhagica.

SYMPTOMATIC PURPURA.

1. *Toxic cases following certain drugs*, such as potassium iodide, chloral hydrate, quinine, copaiba, and more rarely ergot, mercury, and belladonna.

2. *Severe infectious diseases*, such as acute yellow atrophy of the liver, snake-bites, typhoid fever, pneumonia, and the exanthemata.

3. Severe continued *jaundice*.

4. *Profound anæmia, leukæmia, pseudo-leukæmia, scurvy*, and *exhausted and cachectic conditions.*

5. *New-born children with congenital syphilitic change in the arterial walls.*

6. *New-born children without arterial change.* This form occurs in 1 per cent. of children, with a mortality of 75 per cent.

7. *Embolic cases* with malignant endocarditis and with multiple sarcoma.

8. *Neurotic cases*, from vaso-motor relaxation or enfeeblement of the arterial walls, after fright, deep emotion, hysteria, hypnotism, severe neuralgias, and inflammations of the spinal cord.

Symptoms.—Secondary purpura may occur with subcutaneous hemorrhages alone, or with free and visceral hemorrhages as well. Arthritic pains may occur as in purpura rheumatica.

PURPURA RHEUMATICA (PELIOSIS RHEUMATICA, OR SCHÖNLEIN'S DISEASE).

This affection is most common between the ages of twenty and thirty, and is more frequent in males than in females. An antecedent history of rheumatic fever is frequently obtained, but the exact relationship of the disease to rheumatism has not been determined satisfactorily.

Symptoms.—The disease usually begins with a sore throat, malaise, moderate fever, and pain in the joints. The gums are not affected as in scurvy. The articular pain and swelling are due to small hemorrhages in and about the joints. More rarely arthritis with serous or hemorrhagic effusion occurs. Purpura precedes or accompanies the articular pains, and there are frequently associated urticarial wheals which may be hemorrhagic, or any of the manifestations of erythema. Hemorrhagic pemphigus may also occur. The urine may contain albumin.

The **duration** of the disease is between ten days and three weeks. Relapses are common, especially if the patient walk too soon.

The **prognosis** is perfectly good.

PURPURA HÆMORRHAGICA (MORBUS MACULOSUS, OR WERLHOFF'S DISEASE).

Under this heading are included cases of purpura having a disposition to bleed from the mucous membranes and into internal viscera. Severe cases of secondary purpura may be thus included. In some cases purpura hæmorrhagica runs a course more like that of an infectious disease. An acute and a subacute form may be described.

ACUTE PURPURA HÆMORRHAGICA (FULMINATING PURPURA).—The acute form is more common in young adults, but is a rare condition.

1. In some cases the patient is seized with a chill, fever rising to 103° or 104° F., and intense prostration. Purpuric spots rapidly appear, and bleeding occurs from any of the mucous membranes. The patient passes into stupor alternating with restlessness and mild delirum, and dies, either from the hemorrhages or in coma, in from one to seven days. The *prognosis* is bad, 75 per cent. of all cases terminating fatally.

2. In other cases the visceral hemorrhages are the excit-

ing cause of death. Cerebral and meningeal hemorrhages are usually multiple and show no favorite seats of selection. Hemorrhage into the suprarenal capsules may cause death in collapse within a few hours.

3. When the disease attacks pregnant women, miscarriage and severe post-partum hemorrhage result, and the disease runs a rapid, and usually a fatal, course.

Symptoms.—SUBACUTE PURPURA HÆMORRHAGICA.—*Prodromal symptoms* may precede the actual onset; these symptoms comprise malaise, chilly feelings, and a slight evening rise in temperature.

Constitutional Symptoms.—An initial chill may occur, but usually chilly feelings are scattered throughout the course of the disease. The temperature varies from 100° to 104° F., and is higher in severe cases and in children. Prostration is a marked and constant symptom, and usually persists during convalescence. In severe cases the "typhoid condition" may be developed, and in this condition the patient may die. The spleen and the liver are usually enlarged during the attack, and a light form of jaundice is not uncommon. The blood rapidly shows the changes of anæmia.

Hemorrhagic Symptoms.—Purpura develops, the hemorrhagic areas varying from pin-head size to that of the palm of the hand. Extensive ecchymoses may be followed by gangrene of the skin. Free hemorrhages occur from any of the mucous membranes, the most frequent sources of bleeding being respectively the nose, the kidney, the intestines, and the uterus. These hemorrhages may be moderate, or they may be so profuse as to induce a fatal anæmia. Internal hemorrhages into the brain and its membranes, the lungs, or the adrenals may occur, but they are not as common as in the acute form. Pain and swelling of the joints, especially of the hands, the feet, and the knees, may be observed. The articular symptoms are identical with those seen in peliosis rheumatica. In rare cases ankylosis or arthritis may develop. The gums may be normal, or they may be swollen and may bleed, but the teeth are not loosened as in scurvy.

The **pathology** of the disease is unknown. Letzerich has described a bacillus which grows in the liver and enters the blood-vessels, causing in the smaller vessels hyaline thrombi which so weaken the internal wall as to allow of hemorrhage; but his experiments have not been sufficiently verified.

The **duration** of the disease varies from several days to several weeks, but by relapses the disease may be protracted for months or even for years.

The **prognosis** is usually good, but death may result from anæmia, from fatty degeneration of the heart, from exhaustion, or from visceral hemorrhages.

Henoch's Disease.—A severe form of subacute purpura hæmorrhagica has been described by Henoch and bears his name. This form occurs especially in children between the ninth and twelfth years, although it has occurred between the ages of three and forty-six. Males are affected five times as frequently as females. There is a prodromal period with malaise, slight fever, and pain in the joints. The onset is characterized by purpura, pain and swelling of the joints, and severe gastro-intestinal symptoms. These latter symptoms consist of abdominal tenderness with a colicky pain of great severity. The abdomen is rigid and retracted. Rectal tenesmus occurs, with bloody stools. Vomiting is severe, and the vomited matters may contain blood. These gastro-intestinal symptoms seem to be due to hemorrhages in the submucosa or to hemorrhagic infarcts of the small blood-vessels of the intestinal wall. Intestinal ulceration, perforation, and peritonitis may result. The spleen is enlarged. The temperature is slightly raised. Hæmaturia occurs in one-fifth of the cases. These symptoms continue for a few days and then subside, but relapses are the rule, and as many as twenty subsequent attacks have been described. The nature of the disease is unknown.

The **prognosis** is fairly good, being better in children (5 per cent. mortality) than in adults, of whom 25 per cent. die.

Treatment of Purpuric Diseases.

In the secondary purpuras the treatment should be directed toward the exciting causes. In the other forms the treatment is supporting and symptomatic. Arsenic in full doses is at times of service. Iron is indicated for anæmic conditions, but it should be withheld during the acute attacks, as it seems to increase the liability to hemorrhage. In all cases fresh air, good food, and a tonic supporting treatment are indicated. The salicylates are at times of service in peliosis rheumatica, but in some cases they seem useless.

For the hemorrhages various drugs may be used, but no one hemostatic can be relied upon. Among the drugs used are aromatic sulphuric acid, turpentine, acetate of lead, and gallic acid. Epistaxis may require plugging of the nares.

Menorrhagia may be controlled by firm tamponage.

In acute purpura free stimulation is necessary, and in case of profuse hemorrhage rectal or hypodermic injections of sterilized saline solutions should be employed.

In subacute purpura much benefit may be derived from a change of climate, inland places where the air is dry and bracing being preferable.

HÆMOPHILIA.

Etiology.—By "hæmophilia" is meant a constitutional inherited tendency to uncontrollable bleeding. The disease appears in males in the proportion of 13 : 1. Females rarely suffer, although the female members of a bleeding family transmit the tendency to their male offspring. Paternal transmission is exceedingly rare. In rare instances the tendency is acquired.

Pathology.—The exact nature of the disease is unknown. An unusual thinness of the arterial walls has been observed, but this condition is inconstant. The joints may be found to be the seat of hemorrhages, and inflammation of the synovial capsule has been described in a few instances.

The **symptoms** generally appear in early childhood, although excessive bleeding does not usually accompany the cutting of the umbilical cord. The symptoms consist of hemorrhages and inflammation of the joints. The hemorrhages may be spontaneous or may follow traumatism. Cuts or abrasions bleed profusely, and continuous capillary oozing may continue for days after the extraction of teeth. Epistaxis is commonly profuse. These hemorrhages weaken the patient and may at any time prove fatal. Subcutaneous hemorrhages evince themselves as purpuric spots, ecchymoses, and hæmatomata. Large ecchymoses may be succeeded by gangrene. In the female menstruation may be profuse, but parturition is rarely complicated by hemorrhage. The joint-symptoms usually occur after exposure to cold, to which hæmophilic patients are exceedingly susceptible. Pain and swelling occur, especially in the larger joints, and the condition may closely resemble rheumatism.

Prognosis.—Hæmophilia is a contant menace to life. Half the cases die before the seventh year, and only one-eighth reach majority. The younger the patient the more serious the prognosis. The prognosis is better in girls than in boys. Although a serious condition, hæmophilia is not inconsistent, in some instances, with a prolonged and busy life.

Treatment.—Sons born to female members of bleeding families should be protected from external injuries, and the system should be fortified by fresh air and general hygiene. Surgical operations, however slight, should be resorted to only when absolutely indicated, and every appliance should be at hand to check hemorrhage. The hemorrhages, when they occur, should be treated by compression and by the application of the well-known hæmostatic remedies. For the joint-affections rest and soothing applications are indicated.

VII. DISEASES OF THE BLOOD AND THE LYMPHATIC GLANDS.

ANÆMIA.

Anæmia is a generic name applied to deficiencies in the quality or quantity of the blood or of its important constituents. The quantity of the blood may be diminished (oligæmia), or the number of the red corpuscles may alone be diminished (oligocythæmia). In other cases such important constituents as albumin or hæmoglobin may suffer diminution (oligochromæmia). The following classification of anæmia is generally adopted: 1. Secondary anæmia; 2. Chlorosis; 3. Pernicious anæmia.

Secondary Anæmia.

Etiology.—The causes of secondary anæmia have been conveniently classified by Osler as follows:

1. *Anæmia from hemorrhage.*

2. *Long-continued drain on the albuminous materials of the blood*, as in chronic suppuration, Bright's disease, prolonged lactation, or rapid-growing tumors, as cancer.

3. *Anæmia from inanition and defective nutrition*, as from insufficient or improper food, digestive disturbances causing malassimilation, improper modes of life, and intestinal parasites.

4. *Toxic anæmia* results from the use of certain drugs, as lead, mercury, arsenic, salicylic acid, or from organic poisoning, as in syphilis, malaria, infectious diseases, tuberculosis, and pyrexia.

Pathology.—In secondary anæmia the number of the red corpuscles and the percentage of hæmoglobin are proportionately diminished. In severe forms some of the cells may be unnaturally small (microcytes) or of irregular sizes (poikilocytes). Nucleated red cells are always found, although their number may be small in the mildest grades of anæmia. In anæmia after hemorrhage a primary increase in the number of the white corpuscles is usually noticed. The restoration of the watery, saline, and albuminous materials rapidly occurs by absorption, but the regeneration of the red cells is a slower process. Restoration of the normal percentage of hæmoglobin is the last process of regeneration.

General Symptomatology of Anæmia.—1. *Pallor* of the skin and the mucous membranes. It must be remembered that not all anæmic patients are pale, and that not all pale patients are anæmic. Anæmic pallor is best appreciated by the colorless appearance of the ears. In suspected cases of anæmia more reliance should be placed upon the results obtained by counting the red blood-cells and estimating the percentage of hæmoglobin than upon the appearance of the patient.

2. *Cardiac Symptoms.*—The pulse is rapid and usually of low tension. Occasionally a high-tension pulse is encountered. The heart-action is irritable. Palpitation and syncopal attacks are common. In acute anæmia death from syncope may occur. The heart, being supplied with blood of poor quality, tends to become fatty and loses its muscular tone. Mild grades of dilatation are common. A systolic hæmic murmur may be heard over the pulmonary area and is transmitted upward. This murmur comes and goes, and is often appreciated only while the patient lies down. The origin of the anæmic murmur is obscure. There may be heard at the apex a systolic murmur transmitted to the

axilla. This murmur arises from relative mitral insufficiency occasioned by poor muscular contraction, or from slight dilatation of the left ventricle. A continuous venous hum (the *bruit de diable*) may be heard over the jugular vein on the right side of the neck. In extreme anæmia there is a tendency to thrombus-formation, especially in the femoral vein. Unless thrombus occur in the cerebral sinuses, the condition is not serious.

3. *Dyspeptic symptoms* are rarely absent. The tongue is flabby and coated. The bowels are constipated; the appetite is irregular and capricious.

4. *Pulmonary Symptoms.*—Dyspnœa on exertion is in proportion to the extent of the anæmia and the rapidity of its development. In acute anæmia from hemorrhage there may be "air-hunger." A slight cough without expectoration not infrequently occurs.

5. *Cerebral Symptoms.*—There is regularly mental apathy and loss of the power of concentrating the mind. Spots before the eyes, buzzing noises in the ears, and vertigo indicate cerebral anæmia, whether of general or local origin. Headaches, usually more marked in the top of the head and increased by standing, are frequent, but other forms of headache due to digestive disturbances are commonly encountered.

6. There are body-weakness and lack of endurance. The inability to exercise is often aggravated by the dyspnœa thus induced. Slight œdema of the ankles or the legs is not uncommon. An irregular low temperature may be noted in severe cases. Emaciation does not belong to simple anæmia. If present, some primal cause, as tuberculosis or cancer, should be suspected.

7. Menstruation is often affected. There may be menorrhagia, but, as a rule, the menses become scanty and light-colored or may even cease. Amenorrhœa is of no signifi-

cance and demands no special treatment, as it is nature's method of preventing further drains upon the already impoverished blood.

8. *Nervous and hysterical symptoms* are usually present. The patient becomes irritable and restless, sleepless by night, drowsy by day, and may complain of various nervous symptoms, such as hot and cold flashes, irregular pains, and curious sensations in the skin.

The **diagnosis** of anæmia is rendered positive by the results of blood-examination. For methods of counting the corpuscles and of calculating the percentage of hæmoglobin the reader is referred to books on clinical diagnosis. The diagnosis should never rest with the detection of anæmia, but must extend to the discovery of the cause, to which the blood-condition is secondary.

Treatment.—The primal cause, if possible, should be removed by correcting improper modes of life and controlling digestive errors and constipation. The value of fresh air and sunlight cannot be over-estimated, but it is equally important not to over-fatigue anæmic patients by keeping them walking or exercising all day, as is sometimes done.

The specific drug for anæmia is iron. The special preparation used should not be such as to cause constipation or headache. The preparations recommended are Blaud's pill (gr. v, t. i. d.), tartrate of iron and potassium in 10-grain doses in water and glycerin (Price's English glycerin should be used), citrate of iron and quinine, the pyrophosphate of iron, and the liquor ferro-mangans of Gude or of Dietrich. During the administration of iron the bowels must be moved daily, preferably by salines given in the morning. Should iron not be well borne, arsenic or small doses of bichloride of mercury or of binoxide of manganese may be given. In severe cases rest in bed at the beginning of the treatment is to be recommended.

CHLOROSIS.

Etiology and Symptoms.—This condition is common to women between the ages of fourteen and twenty-four. More rarely the affection is encountered in males at the age of puberty. Blondes are more frequently attacked than brunettes. The disease is especially frequent in over-worked factory-girls who live amid poor hygienic surroundings and who work hard upon insufficient or improper food; but cases among the upper classes are not uncommon. Young female immigrants are often attacked soon after their arrival in America. There seems to be some connection between chlorosis and puberty, as in many cases there is the history of precocious development and the early appearance of the menses; in other cases the menses may be retarded. Sir Andrew Clark attributed chlorosis to a blood-poisoning from the absorption of toxic products from a constipated bowel. In some cases it would seem that chlorosis had a primary nervous origin. Mothers chlorotic in their youth are apt to beget chlorotic daughters. In a few instances chlorosis seems to be due to a congenital lack of development of the arterial system (Virchow). *Synonyms:* Chloro-anæmia; Green sickness.

Pathology.—The essential blood-change consists in the reduction of hæmoglobin. In average cases the hæmoglobin falls to 40 per cent., in severe cases to 20 per cent. The number of the red cells may be normal, although, as a rule, they are considerably reduced, but never to the same proportional extent as the hæmoglobin. In a series of 63 cases reported by Osler the average reduction in the number of red cells was 74 per cent.; the average quantity of the hæmoglobin was 42.3 per cent. Poikilocytes, microcytes, and a small number of nucleated red blood-cells may be seen.

Symptoms.—Anæmic symptoms are constant, especially those of nervous and dyspeptic origin. Amenorrhœa is most commonly observed. The color of the skin is not that of anæmia, but is a pale greenish-yellow that is quite characteristic.

There has been described a gastric type of chlorosis with nausea, vomiting, and epigastric pain as prominent symptoms. There may even be vomiting of blood, so that the case may resemble one of gastric ulcer. The diagnosis in these cases from gastric ulcer is often one of great difficulty, and is rendered more uncertain by the fact that gastric ulcer not uncommonly occurs in chlorotic women.

The appetite is apt to be capricious and is even perverted. Constipation is usually constant and obstinate. Emaciation does not occur. Œdema of the ankles may be noticed, and there may be an irregular fever. The cardiac symptoms of anæmia are usually well marked.

The **prognosis** is good for recovery, but relapses are common, and by them the course of the disease may be prolonged. Relapses may occur even during the third decade of life.

Treatment is usually followed by brilliant results if the patient faithfully carries out the directions. Iron is a specific, and under its use the hæmoglobin increases from 5 to 10 per cent. each week. The patient rapidly improves under its use, and often feels capable of discontinuing the treatment, but it is important to continue treatment until the hæmoglobin is above 90 per cent., as otherwise the patent is apt to relapse. The cure in average cases is obtained by about three months' treatment.

Fresh air, good nourishing food, improved hygiene, and the daily use of laxatives, if needed, are important adjuvants to the medicinal treatment. In severe cases a short rest in bed at the beginning of the treatment is often of incalculable service. In the chlorosis of young immigrants peroxide of manganese or permanganate of potassium in 2-grain doses three times a day may be advantageously combined with the iron.

PERNICIOUS ANÆMIA.

Synonyms.—Essential anæmia; Idiopathic anæmia.

Under the heading "pernicious anæmia" are included cases of anæmia running a progressive course and not due to any evident cause. Severe secondary anæmia, resembling the pernicious form in its clinical features, may follow atrophy of the stomach and certain intestinal parasites, especially the bothriocephalus latus and the ankylostoma duodenale, but it is doubtful whether these secondary cases are to be considered as examples of the true pernicious anæmia.

Etiology.—The disease is one of adult life; more rarely it attacks children. Both sexes are equally affected. In some cases there is a history of pregnancy or of parturition; in other cases no assignable cause for the anæmia can be found.

Pathology.—The essential lesions are found in the blood, the liver, and the bone-marrow.

The *blood* is diminished in quantity and is pale and watery. The number of the red blood-corpuscles is greatly reduced, in some instances to as low as one-tenth or less of their normal number (500,000 to the cubic millimeter is not an uncommon reduction; in one instance the number was reduced from the normal 5,000,000 to 143,000). The percentage of hæmoglobin may be reduced in proportion to the reduction in the number of the red cells, or it may even be relatively increased. It is pathognomonic of pernicious anæmia that each red corpuscle remaining in the blood carries its normal, or even more than its normal, load of hæmoglobin. Large and small red corpuscles are seen in the freshly drawn blood ("megalocytes" and "microcytes"), and the corpuscles may be deformed, flask-shaped, and distorted ("poikilocytes"). Nucleated red cells are constantly present, and, if present in large numbers, are distinctive only of pernicious anæmia and of the last stages of leukæmia. In dried and stained specimens of blood two varieties of nucleated cells are seen—one normal in size, with a sharply defined nucleus ("normoblast"), and others of large size, with large, poorly-stained nuclei ("gigantoblasts"). The leucocytes are generally diminished in number.

The *liver* may be enlarged and fatty. The peripheral zones of the acini are pigmented by iron—a condition, in all probability, characteristic of pernicious anæmia.

The *bone-marrow* shows an increase of lymphoid and nucleated red cells, and resembles the red marrow of the child.

There is found fatty degeneration of the heart, the kidneys, and the intima of the smaller blood-vessels. The spleen may be normal or slightly enlarged, and may be pigmented by iron. The lymphatic glands may resemble spleen-pulp in consistency and color. Hemorrhages are usually found under the skin and the mucous and serous membranes.

Two theories of pernicious anæmia have been advanced:

1. Hunter maintains that, by reason of faulty gastro-intestinal digestion, toxic products gain access to the liver and cause extensive blood-destruction, with the deposit of pigment in the liver and the passage of urobilin by the kidneys. Hunter's views, although not absolutely proven, are those generally adopted.

2. The second theory is that there is an increased tenderness or vunerability of the blood-corpuscles, from faulty processes in blood-manufacture.

The **symptoms** are those of progressive anæmia. The color of the patient is a peculiar waxy white or pale lemon. The fat is usually well preserved, and the patient presents a bloated appearance. Syncopal attacks are frequent, and fatal syncope, from fatty degeneration of the heart, may occur. In some cases capillary pulsation and visible pulsation of the arteries may be as well marked as in aortic regurgitation. Hæmic murmurs and slight dilatation of the left ventricle are almost constant. An irregular temperature develops from time to time—usually 100° or 101° F., more rarely from 102° to 104° F. At other times the temperature may be subnormal.

The urine is suggestive of the disease; it is of low specific gravity and of high color, and it contains an excess of urobilin. The pigmentation of the urine, however, is not constant. Gastro-intestinal symptoms of anæmia are common; diarrhœa, however, may not be infrequent. Dropsical swelling of the ankles attends the later stages of the disease, and the dropsy may become general. The tendency to hemorrhage is seen in purpuric spots and in submucous ecchymoses. Retinal hemorrhage is not uncommon. Free hemorrhages from mucous surfaces, with the exception of epistaxis, are rather infrequent.

Prognosis.—The course of the disease is progressive, with periods of temporary improvement, but cases of apparent recovery are not uncommon since the inauguration of the arsenic treatment. Death is usually preceded by a prolonged state of prostration, stupor, and mild delirium with irregular fever.

Diagnosis.—According to Osler, the following are the essential points: (1) The severe grade of reduction in the number of the red cells; (2) their relative richness in hæmoglobin; (3) the presence of many megalocytes and gigantoblasts; (4) the absence of any cause for secondary anæmia; (5) occasional febrile disturbances; (6) the yellow tint of the skin; (7) hemorrhages, particularly retinal; (8) a progressive course and the inefficiency of treatment.

Treatment.—Iron in pernicious anæmia seems to be worthless. The main reliance must be placed on arsenic in full doses, given to the point of tolerance. Osler's plan is to give Fowler's solution in 3-minim doses after meals, increased to 5 minims at the end of the first week, to 10 minims at the end of the second week, and so on until the patient is taking 20 or 25 minims after each meal. Toxic symptoms are rare. Should they occur, the drug is to be discontinued until the poisoning symptoms cease, and is then to be resumed at the dose at which the patient left off. In some cases the addition of phosphorus seems to be beneficial. Iron may be given if arsenic disagrees, but not much is to be expected from it. Rest in bed from time to time is important in conserving the patient's strength. Prolonged residence in a warm inland climate has been recommended, but the climatic treatment is often disappointing. The diet should be light and nutritious. Massage is sometimes found to be beneficial.

Delafield describes a clinical set of cases midway between simple and pernicious anæmia, occurring in those past middle life. The etiology of these cases is obscure. The blood shows the changes only of secondary anæmia. The symptoms are those of a fairly marked anæmia, but improvement under treatment reaches only to a certain degree, and the patients relapse as soon as treatment is discontinued. Absolute recovery does not occur. In this class of cases arsenic seems to be of no use. Iron is the drug on which reliance is to be placed, but dietetic and hygienic treatment seems to be of almost equal service.

LEUCOCYTOSIS.

By the term "leucocytosis" is meant a temporary increase in the number of the white blood-corpuscles; this is a condition entirely distinct from the disease leukæmia. Normally the ratio of white to red corpuscles is 1 : 500, but in leucocytosis the proportion may be 1 : 150 or even 1 : 50.

Physiological leucocytosis occurs during pregnancy and after hearty eating.

Inflammatory leucocytosis occurs in acute infectious diseases attended with local inflammatory reaction. It appears most commonly with pneumonia, diphtheria, and suppurative processes, and it is said to be a sign of good prognostic value.

Cachectic leucocytosis occurs in the cachexias of malignant tumors.

Relative leucocytosis occurs in anæmia, where, from diminution in the number of the red corpuscles, the white cells appear in an increased ratio, although they are not actually increased in number.

LEUKÆMIA (LEUCOCYTHÆMIA).

Etiology.—The cause of the disease is obscure. There occur acute cases which suggest bacterial infection, but upon this point definite knowledge is lacking. In about one-third of the cases there is the history of malarial poisoning. Syphilis seems to possess some obscure relation to the disease. Leukæmia may occur at any age, but it is most common in middle life. Males are affected twice as frequently as females. In women the disease often appears at the time of the climacteric or after pregnancy.

Pathology.—The essential lesions are found in the blood, the spleen, the lymphatic glands, and the bone-marrow. The blood-changes are constant. According to the relative intensity of the changes in the other structures mentioned, splenic (or lienteric), lymphatic, and myelogenous forms have been described. As true myelogenous leukæmia is so very rare, the disease is usually described under two principal forms, (1) splenic-myelogenous or lieno-myelogenous, and (2) lymphatic leukæmia.

Blood-changes consist in the increased number of white cells, their proportion to the red corpuscles rising to 1 : 20 or 1 : 5, or the cells even being in equal proportions. The increased proportion is greater in the splenic-myelogenous form than in lymphatic leukæmia. The blood is pale and watery and may be whitish or brownish-red in color. The red cells are diminished, but not to an excessive degree; hæmoglobin is reduced to a somewhat greater proportion. Nucleated red cells may be seen. Charcot's octahedral crystals separate when blood-slides are kept for some time.

A more detailed account of the changes of the white cells is deemed advisable.

In the normal blood, Ehrlich describes the following varieties of white cells:

1. *Lymphocytes*, small, equal in size to a red corpuscle. The nucleus is large, round, stains deeply, and is surrounded by a narrow rim of non-granular protoplasm.

2. *Large mononuclear leucocytes*, several times larger than the red cells. The nucleus is oval or elliptical and is surrounded by a wide margin of non-granular protoplasm.

3. *A transition form* resembling the preceding, but the nucleus is indentated.

4. *Polynuclear leucocytes*, smaller than the large mononuclear forms, with long, twisted nuclei which stain deeply. The protoplasm is granular and does not stain easily. To these cells, owing to peculiarities of staining, the name "neutrophiles" is given.

5. Cells like the preceding, but the protoplasm contains coarse granules which stain deeply with eosin, hence the name "eosinophiles."

In normal blood these varieties of white cells bear a fixed proportion to each other—the lymphocytes, from 15 to 30 per cent., the polynuclear leucocytes, from 65 to 80 per cent., the mononuclear and transitional forms, 6 per cent., the eosinophiles, from 2 to 4 per cent. According to Osler, the character of the cells in splenic-myelogenous leukæmia differs materially from that in the lymphatic form.

In *splenic-myelogenous leukæmia* the lymphocytes are rarely, if at all, increased; the eosinophiles are present in normal or increased proportion, so that there is a great total increase. The polynuclear neutrophiles are usually relatively diminished. In this form there appears a new variety of cell, derived from the marrow of the bones, and known as the myelocyte. These cells are large and contain a single nucleus, but the protoplasm is finely granular and does not stain well with acid coloring matters, resembling in this regard the neutrophiles.

In *lymphatic leukæmia* the ratio of white to red corpuscles rarely exceeds 1 : 10. The increased number of colorless corpuscles is due to the lymphocytes, which may form 93 per cent. of the total number of white cells. Eosinophiles and red nucleated cells are rare, and myelocytes do not occur.

In mixed forms of leukæmia the blood-condition may deviate from either of these classical types.

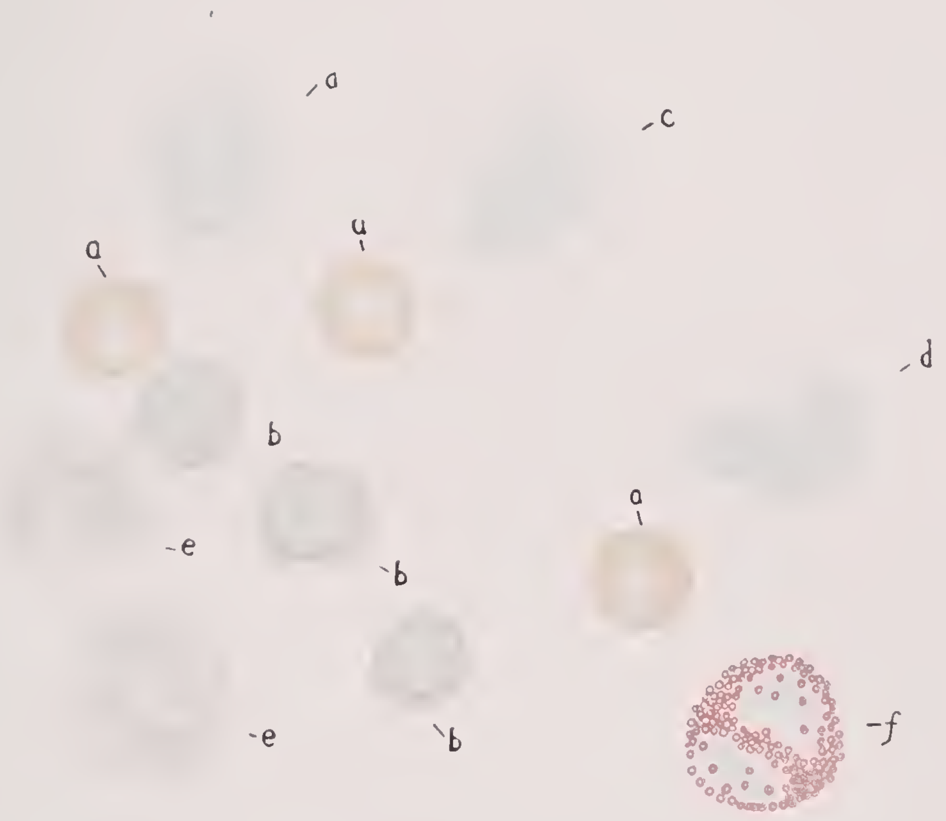
The accurate study of stained blood-specimens is highly important for diagnostic purposes in obscure cases.

The *spleen* is usually much increased in size, weighing from two to eighteen and a half pounds. The enlargement is due to a true hypertrophy of all its constituents. In acute cases the spleen is soft and may even rupture. In protracted cases the organ becomes firmer and the capsule is often thickened and adherent to surrounding structures. On section hemorrhages may be found in its substance, and there may be seen grayish-white areas which consist of aggregations of lymphoid cells.

The lesions in the *bone-marrow* are usually associated with the splenic enlargement. A pure form of medullary or myelogenous leukæmia is exceedingly rare. The marrow, which is yellowish or even purulent in appearance, contains many lymphoid and nucleated red blood-cells.

The *lymphatic glands* may be involved alone (lymphatic leukæmia) or in connection with the splenic and medullary lesions. The histological change consists of hyperplasia of the glandular tissue. The glands are enlarged and soft, but are freely movable and do not mat together. In many cases

LEUKÆMIA. PLATE 22.



Blood stained with Ehrlich's "triple stain" of acid fuchsin, methyl-green, and orange-G; drawn with the camera lucida from normal blood (Osler, in *American Text-Book of the Theory and Practice of Medicine*): *a*, red corpuscles; *b*, lymphocytes; *c*, large mononuclear leucocytes; *d*, transitional forms; *e*, neutrophilic leucocytes with polymorphous nuclei (polynuclear neutrophiles); *f*, eosinophilic leucocytes.

the glands remain perfectly normal. The liver is usually increased in size, from a diffuse infiltration of lymphoid cells. New growths composed of lymphoid cells may be found in various parts of the body, especially in the tonsils, intestinal glands, liver, kidney, retina, lungs, and pleura. The hemorrhagic tendency of the disease is shown by hemorrhages in various parts of the body, especially under the skin, under the serous and mucous membranes, and in the retina.

Symptoms.—1. There are regularly present anæmic symptoms resembling those of pernicious anæmia, so that a detailed description is not necessary. Nervous symptoms, however, are not usually marked. Œdema of the feet and general dropsy are commonly present.

2. Hemorrhagic symptoms may be slight or may lead to a fatal issue. Hemorrhages may occur from any of the mucous surfaces or into the retina, while death may result from cerebral hemorrhages. The most frequent hemorrhages arise from the nose, stomach, intestines, lungs, and kidney.

3. There may be an irregular fever as in pernicious anæmia. Rare cases of acute leukæmia are encountered, in which a continuous temperature of 103° or 104° F. is observed. These acute cases may be mistaken for typhoid fever unless blood-examination be made.

4. Splenic tumor is readily appreciated by palpation. The spleen may extend to or beyond the umbilicus. The free edge is sharp and notched. In acute cases there may be pain and tenderness over the spleen, so that, in connection with its enlargement, abscess of the organ may be suggested. Rupture of the spleen in acute cases has occurred. In protracted cases no symptoms are caused by the splenic tumor except those due to its increased size and weight.

5. Changes in the marrow rarely give rise to clinical symptoms. Exceptionally there is pain in the bones. If the sternum be affected, it may be tender on pressure.

6. Lymphatic enlargements do not occur in all cases. More rarely the lymph-glands are affected alone, with the changes in the blood; to this form the term "lymphatic leukæmia" has been given. These cases usually run a rapid course with fever or hemorrhages. The glands enlarge, but seldom present the same large bunches as in Hodgkin's disease. Pain is rare, the glands usually giving rise to no symptoms except those caused by pressure. The superficial glands are usually first involved, next in frequency the retroperitoneal and mesenteric glands. Enlargement of the abdominal glands may often be appreciated by palpation.

7. Leukæmic new growths may be discovered in the tonsils, retina, or liver. Leukæmic deposits in the liver may give the symptoms of peritonitis.

8. In males persistent priapism may occur; it may be the first symptom of the disease.

9. The urine may be albuminous; uric acid is usually increased.

The **diagnosis** is to be made upon the results of blood-examination. Mild degrees of leukæmia may be differentiated from excessive leucocytosis by the fact that in the latter the polynuclear neutrophiles are alone increased. The diagnosis from Hodgkin's disease can readily be made by the blood-examination.

Prognosis.—The course of the disease is progressive, although through appropriate treatment there may occur periods of temporary improvement. Acute cases terminate fatally within a few months; the less rapid forms terminate at the expiration of one or two years. Death may be due to anæmia, exhaustion, fatty degeneration of the heart, or hemorrhage.

The **treatment** is practically that of pernicious anæmia. Iron may be given, but any great amount of improvement from its use is not to be expected. Quinine should be given to malarial cases, and potassium iodide and mercury should be ordered if the patient be syphilitic. Arsenic is the drug usually employed, and its use is frequently followed by brilliant although temporary results; to be of service, however, the drug must be pushed with due precaution until large doses are reached. During treatment by arsenic the number of the white cells may be much reduced.

Splenic remedies have been tried, but they are not serviceable. Faradism, injections of ergotine and of quinine, and the use of piperin and of oil of eucalyptus have been recommended. Extirpation of the spleen has been performed, with a mortality of 95 per cent. Surgical operations are extremely dangerous in leukæmia, owing to the liability of uncontrollable hemorrhage.

PSEUDO-LEUKÆMIA.

Etiology and Synonyms.—The cause of pseudo-leukæmia is obscure, but of late the view is gaining ground that the condition is an infectious process, and that pseudo-leukæmia really should be assigned to the group of infectious tumors. The disease is more frequent in males than in females, in the proportion of 3 : 1. It occurs at any age, but two-thirds of the cases are observed in those under forty years. In some cases the development of the disease has been preceded by inflammatory conditions of any group of lymphatic glands. *Synonyms:* Hodgkin's disease; Lymphatic anæmia; Adenia; General lymphadenoma; Pseudo-leucocythæmia; Malignant lympho-sarcoma.

Pathology.—The lymphatic glands undergo enlargement. Histologically the process consists of an increase of the lymphoid cells, with or without increase of the reticulum. At first the glands are soft and elastic; they may become firm and hard. Isolated and freely movable at first, the glands tend finally to become fused together to form large lobulated tumors surrounded by a fibrous capsule. The new growth of lymphatic tissue may even extend beyond the capsule to involve neighboring structures. Suppuration of the superficial glands is not uncommon. The glands are usually affected in the following order of frequency; the cervical, axillary, inguinal, mediastinal, retroperitoneal, and mesenteric groups.

The spleen is enlarged in three-fourths of the cases, but the increase is rarely so marked as in leukæmia; in one-half the cases grayish-white tumors are found in its substance; they consist of lymphoid cells and a connective-tissue reticulum. The lymphoid cells of the bone-marrow may be increased in number, often to such an extent that the marrow resembles pus in its appearance.

Secondary growths of lymphatic tissue may occur in any part of the body, especially in the tonsils, in the lymphoid tissue at the base of the tongue, in the liver, spleen, kidneys, lungs, pleura (causing fibrino-serous effusion), spinal cord (causing paraplegia), and in the skin. The ovaries, testicles, and dura mater may also be the seat of new lymphatic growths.

The blood shows the regular changes of anæmia. Poikilocytes and nucleated red cells are not present to any considerable extent. *Leucocytosis does not normally occur* in pseudo-leukæmia, but cases occur, occupying a middle ground between Hodgkin's disease and lymphatic leukæmia, in which the white cells are increased in number and in which the lesions of a pure lymphatic leukæmia may ultimately develop. Some of these cases occur in children under two years of age.

Symptoms are due (1) to anæmia, (2) to the enlargement of the lymphatic glands, and (3) to the secondary lymphatic growths.

1. The *anæmia* gives rise to the regular symptoms of that condition. The pallor, hemorrhages, œdema, dyspnœa, and the cardiac and cerebral symptoms are like those seen in pernicious anæmia, and need not be again described. Pigmentation of the urine, however, does not occur in Hodgkin's disease. The anæmic symptoms may precede or follow those due to the glandular enlargements. The temperature is usually irregularly elevated even during the earlier stages of the disease. The fever may be continuous or recurrent. Remarkable ague-like paroxysms may occur, separated by periods of normal temperature. The duration of the febrile paroxysms may be weeks or months.

2. The *glandular swellings* are frequently the first symptoms noticed. The glands on one side may be involved alone, but later the swellings become symmetrical. At first it may be impossible to exclude syphilitic or tubercular disease of the glands; but later, when large bunched tumors form, the diagnosis is usually easy. Marked variations in the size of the glands may be observed from time to time. In the latter stages the skin may be involved and ulcerated. Enlarged retroperitoneal glands may reach such a size that large abdominal tumors are formed. Besides the disfigurement caused by the glandular tumors, important symptoms may arise from their pressure on neighboring structures. The cervical glands may press upon the trachea and may necessitate tracheotomy. Enlargement of the mediastinal glands may cause pressure upon the trachea, œsophagus, bronchi, vena cava, and aorta. Alarming cardiac disturbance may arise from compression of the vagus. The enlargement of the abdominal glands may cause abdominal pain; they may press upon the portal vein (causing acites and portal obstruction) or upon the common bile-duct (causing persistent obstructive jaundice). Pressure upon the adrenals or upon the splanchnic nerves may be followed by bronzing

of the skin. The axillary glands may press upon the brachial or axillary veins (causing swelling of the arm) or upon the brachial plexus (causing numbness, tingling, pain along the course of the nerves, or paralysis). The enlarged glands in the pelvic and iliac regions may cause sciatic pain and swelling of the leg. Lymphatic growths in the liver and the spleen are regularly followed by an increase in size of these organs.

3. New lymphatic growths in other parts are followed by the regular symptoms of small tumors in the affected structures.

The **diagnosis** from leukæmia is to be made by the blood-examination, there being in Hodgkin's disease no increase in the number of the white cells. The occasional merging of pseudo-leukæmia into lymphatic leukæmia should not, however, be forgotten. The diagnosis from tubercular adenitis is usually rendered easy by the presence of other foci of tubercular disease in the latter condition. Tubercular adenitis usually involves the submaxillary glands, whereas in Hodgkin's disease the glands along the borders of the sterno-mastoid muscle are the glands first to be involved. Suppuration is common to tubercular glands, uncommon to those of Hodgkin's disease.

Prognosis.—With rare exceptions Hodgkin's disease ultimately ends fatally. The course of the disease is frequently marked, however, by more or less prolonged periods of improvement. Acute cases may run a course of several months; the more protracted cases may extend over two or three years. Death results from debility and anæmia, from hemorrhage, from the mechanical pressure of the lymphatic tumors, or from intercurrent disease.

Treatment is mainly that of pernicious anæmia. Arsenic is to be given until the point of tolerance is reached. Phosphorus has also been recommended, but it is of doubtful utility. The internal use of Lugol's solution of iodine in from 5- to 10-drop doses after meals has been recommended. In early cases, in which only a few glands are enlarged, these glands may be removed by surgical operation. Removal of the glands may also be resorted to in case of severe pressure-symptoms.

ADDISON'S DISEASE.

Etiology.—Addison's disease is more frequent in men than in women, and usually occurs in middle life, although no age is exempt. About the causation of the disease there is much doubt. Two theories exist: (1) That the disease is due to loss of function of the suprarenal capsules. (2) That the disease is due to irritation of the abdominal sympathetic plexus, usually owing to disease of the nerves, the ganglia, or the adrenals. In other cases a functional nerve-disturbance must be supposed to exist.

Pathology.—In 88 per cent. of all cases the adrenals are found diseased. In the vast majority of cases the lesion in the adrenals is tubercular, the capsules being converted into masses of fibrous tissue and cheesy matter. In other cases the adrenals are found atrophied, absent, or the seat of malignant tumors. Against the theory that Addison's disease is due to loss of function of the suprarenal capsules through disease are the following facts: (1) In 17 per cent. of all cases of Addison's disease the adrenals are found to be normal. (2) Every variety of adrenal lesion has occurred without giving rise to the symptoms of Addison's disease. Owing to improved technique in nerve-staining, there are found an increasing number of cases of Addison's disease in which lesions are found in the sympathetic nerve-structures in the abdomen. Of the 30 cases most recently examined, 27 showed sympathetic nerve-lesions. The ordinary lesions found in the ganglia and nerve-fibres consist of degeneration, congestion, hemorrhages, and infiltration by leucocytes or new connective tissue. The blood shows the changes common to anæmia. The heart may be fatty; it is seldom enlarged. The liver shows no essential lesion. The spleen may be somewhat increased in size. Tubercular changes are often found in various parts of the body if tubercular disease of the adrenals be present.

Symptoms.—Four cardinal groups of symptoms appear: (1) prostration, (2) heart weakness, (3) gastro-intestinal symptoms, and (4) bronzing of the skin.

1. *Prostration* is shown by the early appearance of intense languor both of body and of mind. The patient becomes weak, dull, apathetic, listless, and peevish. The symptoms of prostration are constant and progressive.

2. *Heart weakness* is attended by frequent syncopal attacks, any one of which may be fatal. The pulse is feeble and rapid. The poor condition of the circulation induces the symptoms of cerebral anæmia, which usually are well marked.

3. *Gastro-intestinal symptoms* are almost constant. Nausea and vomiting may, with the prostration, appear as initial symptoms. The vomiting, which is not usually influenced by diet, cannot be accounted for by any lesion found in the stomach, but seems to be of nervous origin. The vomiting occurs in violent paroxysms and becomes more distressing as the disease progresses. Diarrhœa is twice as frequently observed as constipation.

4. The bronzing of the skin is usually observed after the constitutional symptoms have lasted for some little time. In other cases it is the first symptom observed. The pigmentation of the skin usually begins in the exposed portions of the body, as the face and the hands, or in areas exposed to friction of the clothing, or in places which are normally pigmented, as about the nipples. The color varies from a

yellow to a brown or even a black. The pigmentation may at first occur in scattered areas, but finally tends to become diffused, so that the patient may resemble a mulatto. Similar discoloration may be found in the mucous membrane of the lips, gums, and tongue. Internal pigmentation does not usually occur.

Among the remaining symptoms to be enumerated, the most important are pain and tenderness in the lumbar or epigastric regions; these symptoms occur in one-third of the cases. In the last stages of the disease the patients become extremely feeble and may develop stupor, delirium, coma, or general convulsions. Death occurs from asthenia or from syncope. In a few acute cases the disease runs a course with fever, vomiting, diarrhœa, and intense exhaustion, and before the pigmentation appears the diagnosis may be impossible.

Prognosis.—Recovery is practically unknown. Acute cases may terminate within a few months. The average duration is about one year, but cases are on record in which the symptoms continued as long as ten years.

Treatment is symptomatic, as for the disease itself no curative treatment is known. The patient should be guarded from causes leading to syncope, and anæmic conditions should be controlled by iron and arsenic. The vomiting may be alleviated by bismuth, creosote, hydrocyanic acid, or codeia. Purgatives should be given with caution, as they may induce an exhausting diarrhœa.

TUBERCULOSIS OF THE LYMPH-GLANDS (SCROFULA).

Etiology.—Scrofula is tubercle, and the etiology of scrofula is therefore that of other tubercular infections. Tubercular infection of lymphatic glands is favored by previous adenitis, so that children with catarrhal inflammations of the mucous membranes that excite adenitis of the neighboring lymphatic glands seem to be especially subject to subsequent lymphatic tuberculosis.

The **pathology** of scrofula is that of tubercular foci, which are usually localized in certain groups of glands and show a tendency to spontaneous healing. In many cases suppuration of the infected glands occurs, especially with tubercular glands in the neck. In these instances the pus is usually sterile, and it is not known whether the suppuration is excited by the tubercle bacilli and their products or by a mixed infection of pus-organisms. An unhealed focus of tubercular adenitis may at any time discharge bacilli into the blood-vessels or the lymph-vessels; it is said that three-quarters of the cases of acute miliary tuberculosis originate in this way.

Symptoms.—1. *General tuberculous lymphadenitis* is a rare condition, and usually occurs in the negro race. The lymph-glands throughout the body are the seat of a diffuse tubercular infiltration. Acute cases resemble clinically Hodgkin's disease, but there is apt to be more fever.

2. *Local Tuberculous Lymphadenitis.*—(*a*) *Cervical.*—This form is frequently seen, especially in tenement-house and asylum children and in the negro race. The submaxillary glands are usually the first to be involved, and subsequently the cervical chains of glands become infected. The glands may remain isolated and mobile, but they tend to become fused so as to form large knobby tumors. Suppuration is common. For the details of this affection the reader is referred to works on surgery.

(*b*) *Bronchial.*—The bronchial glands are extremely subject to infection, and they may be involved with or without local lesions in the lung. Acute miliary tuberculosis is apt to result. (For details see Miliary Tuberculosis and Broncho-pneumonia.)

(*c*) *Mesenteric* (*Tabes Mesenterica*).—In this form the mesenteric and retroperitoneal glands are enlarged and tubercular. They may suppurate, or they may become encapsulated and infiltrated by lime-salts. Mesenteric tuberculosis may be primary or may complicate tubercular disease of the intestines.

The **treatment** of tubercular adenitis is that of tuberculosis in general. Cervical tubercular glands may be removed.

VIII. DISEASES OF THE NERVOUS SYSTEM.

1. DISEASES OF THE MEMBRANES OF THE BRAIN.

(*a*) Diseases of the Dura Mater.

ACUTE EXTERNAL PACHYMENINGITIS.

Etiology.—Pachymeningitis is regularly secondary to injuries and diseases of the cranial bones and to suppurative disease of the middle ear and of the mastoid cells.

Pathology.—The dura is thickened by a purulent infiltrate; the products of inflammation collect between the dura and the skull, forming a circumscribed abscess. The lesion is usually localized over one cortex. The inflammation may extend to the pia mater or to the venous sinuses.

The **symptoms** are usually obscure. Pain is usually referred to the seat of the lesion. Septic symptoms develop, and compression-symptoms may result in hemiplegia if the motor area be pressed upon.

The **prognosis** is good if the treatment be scientific and if the pia and the venous sinuses be not involved.

The **treatment** consists in trephining and draining the abscess-cavity.

ACUTE INTERNAL PACHYMENINGITIS.

Etiology.—Pachymeningitis interna is secondary to inflammation of the external surface of the dura, or complicates erysipelas, Bright's disease, pyæmia, pneumonia, puerperal fever, and the exanthemata.

Pathology.—The inner surface of the dura is covered with fibrin and pus or with pus alone; the thickness of the dura is not usually involved. The inflammation is apt to extend to the pia and to the venous sinuses. The purulent exudation is usually circumscribed over the cortex.

The **symptoms** resemble those of a localized purulent meningitis. In the complicating cases the symptoms may be so obscured by those of the primary disease that the diagnosis is rendered obscure.

The **prognosis** is not good, owing to the liability to meningitis and thrombosis of the cerebral sinuses.

The **treatment** is that of meningitis.

CHRONIC INTERNAL PACHYMENINGITIS.

Etiology and Synonyms.—The disease is usually found in males over fifty years of age; it occurs in connection with insanity and degenerative diseases of the brain. Almost all the subjects are markedly alcoholic, and the disease is one almost exclusively of tramps and almshouse inmates. *Synonyms:* Hemorrhagic pachymeningitis; Hæmatoma of the dura mater.

Pathology.—The disease is characterized by the growth of a new membrane upon the dura, usually involving a small area over one cortex. In the *earlier stages* this membrane resembles a brownish-red staining of the dura, and consists of large, thin-walled blood-vessels supported by a delicate connective-tissue framework. From these blood-vessels hemorrhages occur, constituting the principal feature of the disease. In the *later stages* the dura is thickened over the affected area by dense fibrillated connective tissue, and upon its inner surface the original membrane is found as already described. The dura may be from half an inch to an inch thick, and thus the brain becomes slowly compressed; the compression of the brain is further increased by hemorrhage between the dura and the pia, which may occur at any time.

The **symptoms** are due to slow and to sudden brain-compression.

Slow Compression.—Headache is prominent, constant, and usually localized. There are loss of memory and increasing stupidity. The gait is shambling, slow, and unsteady, but paralysis and ataxia are not observed. The speech becomes slow, faltering, and scanning. One or both pupils may be contracted. In the earlier stages of the disease these symptoms are not marked.

Sudden compression is caused by meningeal hemorrhages. The patient will become unconscious, with or without preceding convulsions, and will develop hemiplegia (see Meningeal Hemorrhage). The hemorrhage may occur spontaneously or after exertion, or after a blow or an injury to the head. In the latter instance the disease possesses great medico-legal interest. The hemorrhage may occur early in the disease and may be the initial symptom.

Prognosis.—The disease is slow in its progress, extending over years. Rare cases of recovery have been reported. Death usually occurs from degeneration of the brain with insanity, or from hemorrhage.

Treatment.—As the diagnosis from syphilitic meningitis cannot in all cases be made, potassium iodide should be given in full doses. This treatment, however, is of no service in non-syphilitic pachymeningitis. The treatment of the disease is chiefly prophylactic. Quiet, easy employment, and the avoidance of severe bodily exertion should be enforced, to lessen the chance of meningeal hemorrhage.

SYPHILITIC PACHYMENINGITIS.

This disease will be considered under the heading of Syphilitic Disease of the Brain-membranes.

(*b*) DISEASES OF THE PIA MATER.

The pia mater may be the seat of a tubercular, a non-tubercular, and a syphilitic inflammation. Its inflammations are called "meningitis" or "leptomeningitis."

TUBERCULAR MENINGITIS.

Etiology.—In young children tubercular meningitis is regularly only part of an acute general tuberculosis. For the etiology see the latter disease.

In adults tubercular meningitis is usually a local inflammation secondary to some pre-existing tubercular disease. In some adults with acute general tuberculosis the lesions of tubercular meningitis may be found at the autopsy, but clinical symptoms during life are not usually observed in these cases.

Tubercular meningitis is most common in children between two and seven years of age. In adults the disease is rare after the twenty-fifth year. The attack may seem to be induced by some exciting cause, as a blow, a fall, or exposure to a hot sun.

Pathology.—Upon removing the skull-cap the brain appears too large and the convolutions are flattened. The tubercles appear as small grayish points, occasionally with cheesy centres. The tubercles are most abundantly found along the course of the blood-vessels and in the sulci. They may be confined to the base (hence the name "basilar meningitis") or they may be more uniformly distributed. The pia about the tubercles is thickened by serum and by cellular infiltration. Properly speaking, there should be no purulent exudate, but a fibrino-purulent exudate is not uncommon from a "mixed infection." The cortex is œdematous and is infiltrated with cells. In children the ventricles are regularly distended with clear or turbid serum, and the walls of the ventricles are studded with tubercles ("acute hydrocephalus"). This distention may be so great that the septum between the lateral ventricles will rupture, and great compression of the brain from within outward will ensue. In adults the distention of the ventricles is less frequent. In all cases tubercular inflammations are seen in other parts of the body.

The **symptoms** may be classically divided into four groups:

Prodromal Symptoms.—The child is irritable, restless, and disinclined to play. Causeless vomiting is a symptom which in children is suggestive of incipient meningeal inflammation.

Symptoms of Brain-irritation.—The child may be taken with convulsions or a chill. Fever develops and runs an irregular course. The temperature may vary between 100° and 103° F. with irregular remissions; more rarely a fever of 104° to 105° F. is encountered. The temperature gives no guide to the diagnosis, nor does it indicate the actual severity of the disease. Headache becomes severe, and the special senses are so hyperæsthetic that the child becomes intolerant of light or noise, and the hyperæsthesia of the skin may render any handling of the patient extremely painful. There is rigidity and retraction of the back of the neck; the muscles generally are rigid and resist passive motion, and the general attitude is one of general flexion which is quite characteristic. Convulsive movements or automatic motions are common. Mild or violent delirium develops, alternating with periods of stupor in which the child may moan or give vent to the shrill "hydrocephalic cry." The pupils are usually contracted. The pulse varies between 110 and 120. Vomiting attacks occur without nausea, and are not influenced by the taking of food. The bowels are usually obstinately constipated. The abdomen is retracted and "boat-shaped."

Symptoms of brain-compression next appear, and are due not only to the thickening of the pia, but also to the distention of the ventricles. The child becomes dull and apathetic. The special senses are blunted. Stupor succeeds the delirium and merges into coma. The automatic motions of the hands and feet may continue, or there may be evident paralysis of certain groups of muscles, of which paralyses squint is the most easily recognized. The pulse may now become slowed to 90, 80, or even 60 in the minute. This characteristic slowness of the pulse, however, may not be marked in young children. Irregularity of the pulse is frequently observed. The coma becomes profound, the sphincters relax, paralysis becomes more evident; the pulse during the last stages of the disease becomes rapid and feeble, and the breathing becomes irregular and may assume the Cheyne-Stokes variety. Death occurs from depression of all the vital functions.

Not all cases, however, run this classical course. The symptoms of irritation and of compression may be variously admixed, according to the intensity of the inflammation in various portions of the pia mater. Some young children are seized at the onset with convulsions, which may be repeated; the child becomes stupid and drowsy, moans as if in pain, and develops irregular fever; the rhythm of the respirations becomes disturbed, and coma precedes a fatal issue. In adults, owing to the less frequent movement of the ventricles, the compression-symptoms are less marked.

The **prognosis** is uniformly bad.

The **duration** of the disease is from one to four weeks.

The **treatment** is that of acute non-tubercular meningitis.

ACUTE NON-TUBERCULAR MENINGITIS.

Etiology.—Meningitis is regularly due to germ-infection of the pia mater. The inflammation of the pia mater occurs as the characteristic lesion of epidemic cerebro-spinal meningitis.

Cases of secondary pus-infection occur more commonly, the germs infecting the meninges (1) by direct extension or (2) through the arteries.

1. *By direct extension*, following inflammation or injury of the bones of the skull, of the dura mater, or of the orbital cavity. The most frequent cause is necrosis of the petrous portion of the temporal bone from middle-ear disease. The germs may travel along the nerve-roots during the course of facial erysipelas, or a phlebitis arising from suppuration of the orbit or cheek may infect the cavernous sinus and thus spread to the pia.

2. *Through the Arteries.*—Infectious emboli may occur in the course of pyæmia, abscess of the lung, and malignant endocarditis. Bacterial infection also occurs during the course of certain infectious diseases, especially pneumonia, erysipelas, typhoid fever, rheumatism, and the exanthemata. Infection is favored by Bright's disease and gout. The congestion following sunstroke predisposes to meningitis.

Pathology.—Two anatomical forms are found, which during life give rise to the same clinical symptoms:

1. *Cellular Meningitis.*—The pia is congested, dry, and lustreless. The substance of the pia is the seat of a cellular infiltration. There is no purulent exudate. This form is analogous to the cellular form of peritonitis in peritoneal septicæmia.

2. *Exudative Meningitis.*—There is an exudation of fibrin, serum, and pus into the thickness of the pia, more rarely appearing upon its free surface. In children and in young adults the lining of the ventricles becomes inflamed and the ventricles become distended with turbid serum. The pia mater covering the spinal cord may be involved by direct extension. The locality of the meningitis varies. In

pneumonia and in malignant endocarditis the process is usually bilateral and limited to the cortex; with middle-ear disease the lesion is over the temporo-sphenoidal lobe on one side; in other cases the base alone may be involved. In children, should the patient recover from the meningitis, the ventricles may remain distended for some time.

The **symptoms** depend largely upon the character of the original disease.

1. If the meningitis follow middle-ear disease, the symptoms of meningitis are well developed and resemble those of the tubercular or the epidemic form, so that a further description is unnecessary. The diagnosis from tubercular meningitis is to be made by attention to the following points:

Tubercular Meningitis.	*Simple Meningitis.*
History of tuberculosis.	History of ear disease, etc.
No apparent cause.	Cause evident.
Longer prodromal period.	Short prodromal period.
Longer course.	Short course.
Presence of tuberculosis in the lungs, etc.	No tuberculosis.
Heredity in 20 per cent.	No heredity.

2. If the meningitis complicate severe infectious disease, the symptoms may be obscured by those of the original disease. Retraction of the head and paralyses may constitute the only suggestive symptoms. Facial paralysis and squint are usually the most evident. The pulse may or may not become slower, but it usually becomes irregular. Irregularities of the rhythm of respiration are usually observed. It may be impossible to differentiate between pnuemonia in children with cerebral symptoms and pneumonia complicated by meningitis.

The **duration** is about a week, but the disease may continue for from two to four weeks.

The **prognosis** is bad, yet a number of patients recover.

Treatment.—The patient should be kept free from noise and light. The continual use of the ice-cap throughout the disease is frequently of service, and should always be employed. Leeches should be applied behind the ear in robust cases, but depletion in the latter stages of the disease is not to be advised. Blisters to the occiput add to the discomfort of the patient and do no good. Calomel and magnesium sulphate should be given at the outset, in such doses as will act on the bowels and reduce the meningeal congestion. Ergot is advised during the earlier stages, to reduce congestion. Potassium iodide in 5- to 10-grain doses is employed as a routine treatment, but its use is theoretical. The restlessness and headache are to be controlled by opium, phenacetine, chloral, bromide of sodium, and sulphonal. For the tubercular form the head may be shaved and covered with iodoform ointment; for this treatment good results have been claimed. Should meningitis follow middle-ear disease or suppurative disease of the dura, localized trephining and drainage should be practised.

To avoid meningitis prophylactic treatment should be directed toward the careful cleansing of the ear in suppurative otitis media, the opening and draining of abscesses of the mastoid cells, and the antiseptic treatment of suppurative processes about the cheeks and the orbit.

SYPHILITIC MENINGITIS.

(See *Syphilis of the Brain.*)

CHRONIC MENINGITIS.

Etiology and Synonym.—The disease is one of middle life, and is most commonly seen in those who have led a life of privation and exposure. It is common in tramps and in the inhabitants of almshouses. Chronic alcoholism and cerebral endarteritis seem to lead to the disease. Chronic meningitis may complicate fractures or inflammation of the cranial bones, chronic pachymeningitis, chronic Bright's disease, chronic degenerations of the brain, and slow-growing cerebral tumors. *Synonym:* Chronic leptomeningitis.

Pathology.—The pia mater is thickened, opaque, œdematous, and infiltrated with cells. There may be adhesions between the pia and the dura. The brain-cortex may be softened or sclerotic. The ventricles may be distended with clear serum, and the ependyma lining them may be thickened and rough. The meningitis may be localized at the base or the cortex of the brain.

The **symptoms** are those of slow compression; they resemble those of chronic pachymeningitis except that hemorrhages do not occur.

Prognosis.—The disease is chronic in its course, extending over years, but recovery cannot be expected.

Treatment.—Syphilitic meningitis should be excluded by a conscientious trial of mercury and of potassium iodide in full doses. Otherwise the treatment is symptomatic.

MENINGEAL HEMORRHAGE.

Hemorrhage may occur between the dura mater and the bones of the skull, and between the dura and the pia mater.

HEMORRHAGE BETWEEN THE DURA MATER AND THE BONES OF THE SKULL.—**Etiology.**—These hemorrhages are regularly due to violence, either by concussion separating the dura from the cranial bones and lacerating the middle meningeal artery, or by fracture of the cranial vault.

The **symptoms** are those of shock, laceration and compression of the brain, followed by the symptoms of meningitis. Although these cases are of surgical rather than of medical interest, they are important to the physician, owing to the unpleasant results that follow an erroneous diagnosis. A man whose breath is alcoholic may be found unconscious in the street with a scalp-wound. The case is regarded as one of alcoholism, but the coma deepens, the temperature rises, and the patient dies. At the autopsy there is found a fracture of the skull with laceration of the middle cerebral artery and meningeal hemorrhage.

Treatment.—In suspected cases incision should be made, exposing the site of probable fracture. When the diagnosis is made, trephining and removal of the clots should at once be resorted to.

HEMORRHAGE BETWEEN THE DURA MATER AND THE PIA MATER.—**Etiology.**—This form of meningeal hemorrhage may occur from—(1) Traumatism; (2) thrombosis of the venous sinuses; (3) in new-born children as the result of severe labor or the pressure of forceps; (4) chronic hemorrhagic pachymeningitis; (5) rupture of an aneurysm of one of the cerebral arteries; (6) after convulsions in children; (7) hemorrhagic diseases. The disease may occur at all ages, thus differing from cerebral hemorrhage.

Pathology.—The hemorrhage may be at the base of the brain, at the convexity, or may be more equally distributed. Small hemorrhages may ultimately be absorbed, leaving hæmatin staining. It must be remembered that in cerebral hemorrhage the blood may rupture through the cortex or may leak out by the fourth ventricle and appear between the membranes.

The **symptoms** vary according to the size, location, and cause of the hemorrhage.

1. *Large Clot over One Cortex.*—There is sudden coma, with stertorous breathing, slow pulse, and abolition of all reflexes. There may be hemiplegia or monoplegia, according to the size and position of the hemorrhage. Convulsive movements of muscles ultimately to be paralyzed may occur. The temperature falls to 96° or 97° F., but subsequently

rises to 103° or 105° F. or even higher. The patient may die in coma within twenty-four hours, or may die in several days with the symptoms of meningitis. Recovery occurs only if the clot be small. Small hemorrhages over a convexity may give rise to the symptoms of acute meningitis without the occurrence of sudden coma.

2. *Clots over both hemispheres* give rise to sudden coma and general convulsions, so that the diagnosis from uræmia may be one of great difficulty.

3. *Hemorrhage at the base of the brain* compresses the medulla and leads to death in a few hours. A high antemortem temperature is usually observed in these cases.

4. *Meningeal Hemorrhage of the New-born.*—The child may be stillborn, or it may be born in asphyxia, from which it may die, or from which it may recover, only to die in coma with convulsions within a few days. In those who live, symptoms of paralysis with or without athetosis, mental defects, and epileptic seizures may develop (see Cerebral Atrophy of Children).

The **prognosis** of meningeal hemorrhage is bad unless the clot is small and is situated over the convexity. Recovery may be complicated by permanent paralysis (with or without convulsive movements) of groups of muscles upon the side opposite to the lesion. Death from meningeal hemorrhage usually occurs earlier than from cerebral hemorrhage.

2. DISEASES OF THE BLOOD-VESSELS OF THE BRAIN.

CONGESTION.

Congestion may be active or passive.

Active hyperæmia may be due to exposure to the sun, to the ingestion of such drugs as alcohol, amyl nitrite, and nitroglycerin, to excessive brain-work, to reflex causes, or to fever.

Passive hyperæmia results from (1) mechanical obstruction to the venous return of blood, as with tumors of the neck or strangulation; (2) from general venous congestion due to heart or lung disease.

The **symptoms** are neither characteristic nor constant. The active congestion causes headache, a sense of fulness and throbbing in the head, and hyperæsthesia of the special senses. The face is flushed; the superficial arteries pulsate visibly. Passive hyperæmia gives rise to dull headache, to mental slowness, to disturbances of sleep, and to a feeling of fulness in the head. Attacks of delirium or unconsciousness may attend the severer forms of congestion.

Treatment.—The patient should be kept quiet, and the bowels should be freely moved. Venesection may be indicated in acute congestion of an intense type, and an ice-cap should be applied to the head. In passive hyperæmia the treatment should be directed toward the cause of the condition.

ANÆMIA.

Anæmia may result from general or local causes. Localized anæmia may be due to vaso-motor constriction, endarteritis, or cerebral compression.

Symptoms.—Acute anæmia, such as results from profuse hemorrhage, gives rise to confusion of ideas, marked dyspnœa amounting to "air-hunger," spots before the eyes, ringing noises in the ears, a tendency to yawn, nausea, and dilated pupils. Convulsions and syncope may occur. Sudden death in syncope may result from an intense anæmia suddenly induced.

Chronic anæmia is characterized by vertical headache, disturbances of sleep, lack of mental power and concentration. There are spots before the eyes and buzzing in the ears. There may be repeated syncopal attacks. The symptoms are regularly relieved by lying down.

The **treatment** is that of anæmia.

ŒDEMA.

Œdema may be due (1) to atrophy of the cerebral cortex, there being an increase of the cerebro-spinal fluid and œdema of the overlying pia: to such a condition the term "wet brain" has been applied; (2) to long-continued passive congestion; (3) localized œdema occurs about tumors and abscesses; (4) œdema may occur during the advanced stages of Bright's disease.

The **symptoms** are obscure, and, in general, are due to the disease to which the œdema is secondary.

CEREBRAL HEMORRHAGE (APOPLEXY).

Etiology.—Cerebral hemorrhage usually occurs after the age of forty, the age of the greatest liability being between the seventieth and eightieth years. More rarely the disease occurs in children and in young adults. The condition is more common in males than in females. An "apoplectic habit" has been described—a short, thick-set body, with flushed face, prominent color, and short neck, but, as a matter of fact, hemorrhage is, if anything, more common to those of spare habit. In almost all cases there occurs degeneration of the cerebral arteries, rendering them liable to rupture. Fatty degeneration, atheroma, or the weakening of an artery from an embolus may be found, but the most frequent cause of hemorrhage is an endarteritis which allows of the production of miliary aneurysms, from whose rupture the hemorrhage takes place. These miliary aneurysms occur with greatest frequency upon the middle cerebral artery; they vary in size up to that of a pin's head. The predisposing causes for the endarteritis are gout, syphilis, alcoholism, and a life of over-work and privation. There may be an inherited tendency toward arterial degeneration. The exciting cause for rupture is an increased arterial tension. As high tension so frequently occurs with chronic nephritis and hypertrophy of the left ventricle, the association of these conditions with cerebral hemorrhage is exceedingly common. A sudden increase of blood-tension may occur from strain, fright, anger, a cold bath, or over-eating or drinking. In other cases the hemorrhage may occur during rest or sleep.

Cerebral hemorrhage in young adults may be associated with congenital lack of development of the aorta and large vessels. Hemorrhage may occur during the course of hemorrhagic disease, as leukæmia, pernicious anæmia, and purpura hæmorrhagica, but these hemorrhages are usually multiple and have no especial seat of selection.

Pathology.—Cerebral hemorrhage usually occurs from the middle cerebral artery, and involves the internal capsule, optic thalamus, corpus striatum, and the neighboring brain-tissue. Next in frequency are hemorrhages into the cortex, pons, and cerebellum. The right side is more frequently involved than the left. Should the hemorrhage occur near the cortex, the blood may rupture through and appear beneath the dura. Rupture into a ventricle may also occur. The clot at first is red, soft, and admixed with lacerated brain-tissue. Gradually the clot becomes firmer, the hæmoglobin becomes converted into reddish-brown hæmatoidin, the disintegrated brain-tissue undergoes fatty degeneration and absorption, and a connective-tissue capsule may form about the hemorrhagic mass. Ultimately there may be left a pigmented puckered cicatrix, or a mass of softened pigmented tissue with or without a connective-tissue wall, or a cyst with brownish fluid contents. The torn brain never unites. From the point of laceration secondary degeneration occurs, upward to the cortex if the sensory tract be involved, downward in the motor tract as far as the anterior motor cells of the spinal cord if the lesion involve motor fibres (see Secondary Lateral Sclerosis).

The **symptoms** depend upon the size, rapidity, and position of the hemorrhage. The symptoms are the prodromal, those of the attack, and those of the chronic stage.

Prodromal symptoms, which are due to the cerebral endarteritis, consist of headache, dizziness, ringing in the ears, and irritability of temper. More characteristic are temporary loss of speech, incomplete temporary paralysis of an arm or a leg, or temporary and partial blindness.

Symptoms of the Attack.—The patient, without warning, becomes dizzy and falls unconscious. In other cases coma may be developed gradually, or may be preceded by forgetfulness and mental aberration. The face is flushed; the arteries of the neck pulsate visibly; respirations are slow and stertorous, and may be irregular; the eyes and the head are turned toward the side of the lesion (" conjugate deviation "); the pupils vary, but are usually dilated and inactive; the pulse is full, slow, and of high tension; the temperature at the time of onset falls to subnormal, but within twenty-four hours it begins to rise. The more severe the hemorrhage, the greater is the initial fall and the higher the subsequent rise. Evidences of paralysis may be discovered: the mouth may be drawn from the paralyzed side; the paralyzed cheek may blow out during respiration more than the other cheek, and the naso-labial fold may be obliterated. Hemiplegia may be discovered by finding that the affected arm and leg drop more " dead " than do those of the unparalyzed side, and lack their normal " tone." The urine may contain a trace of albumin or of sugar, even if there be no nephritis.

Small hemorrhages slowly developing may cause no actual coma, but bewilderment and mental confusion are commonly observed in these cases. Hemorrhages in the cortex or into a ventricle may cause convulsions at the onset, but these cases are uncommon. In this condition of coma the patient may die, from involvement of the vital functions or from hypostatic pneumonia and pulmonary œdema.

Symptoms of the Chronic Stage.—When the attack does not prove fatal, consciousness becomes gradually restored and the reflexes return, and it becomes possible to gauge the extent of the damage done by the hemorrhage. The symptoms of motor paralysis depend upon the extent to which the motor tract is lacerated. If the hemorrhage be in its usual location, there remains a hemiplegia of the opposite side. The muscles, however, are not uniformly paralyzed. Those muscles used automatically and in pairs, as the muscles of respiration, escape paralysis; the arm is more paralyzed than the thigh, the hand and foot more than the arm and leg. If recovery ensue, the larger and more automatic muscles improve more rapidly and completely, the leg before the arm, the arm before the hand. An "initial rigidity" of the paralyzed muscles is due to irritation about the lesion. An "early rigidity" may develop on the second or third day and may last for one or two weeks; this rigidity is due to inflammation about the lesion. In early rigidity the position of the limbs is one of rest. "Late rigidity" appears after several weeks and is usually permanent. It is due to descending degeneration of the motor tract (see Secondary Lateral Sclerosis). The position of the affected limb is generally one of flexure. Slow convulsive twitchings may occur in the paralyzed muscles; this "post-hemiplegic chorea" is due to destructive lesions of the optic thalamus. During late rigidity the reflexes of the affected limbs are greatly exaggerated. Atrophy of the paralyzed muscles does not occur. Facial paralysis occurs with hemiplegia in lesions of the internal capsule, but the eyes can be closed. The tongue deviates when protruded toward the paralyzed side. Aphasia of some kind may accompany right hemiplegia or may occur alone. Hemianæsthesia develops after lesions of the posterior portion of the internal capsule, but is seldom absolute or permanent. Mental symptoms attend

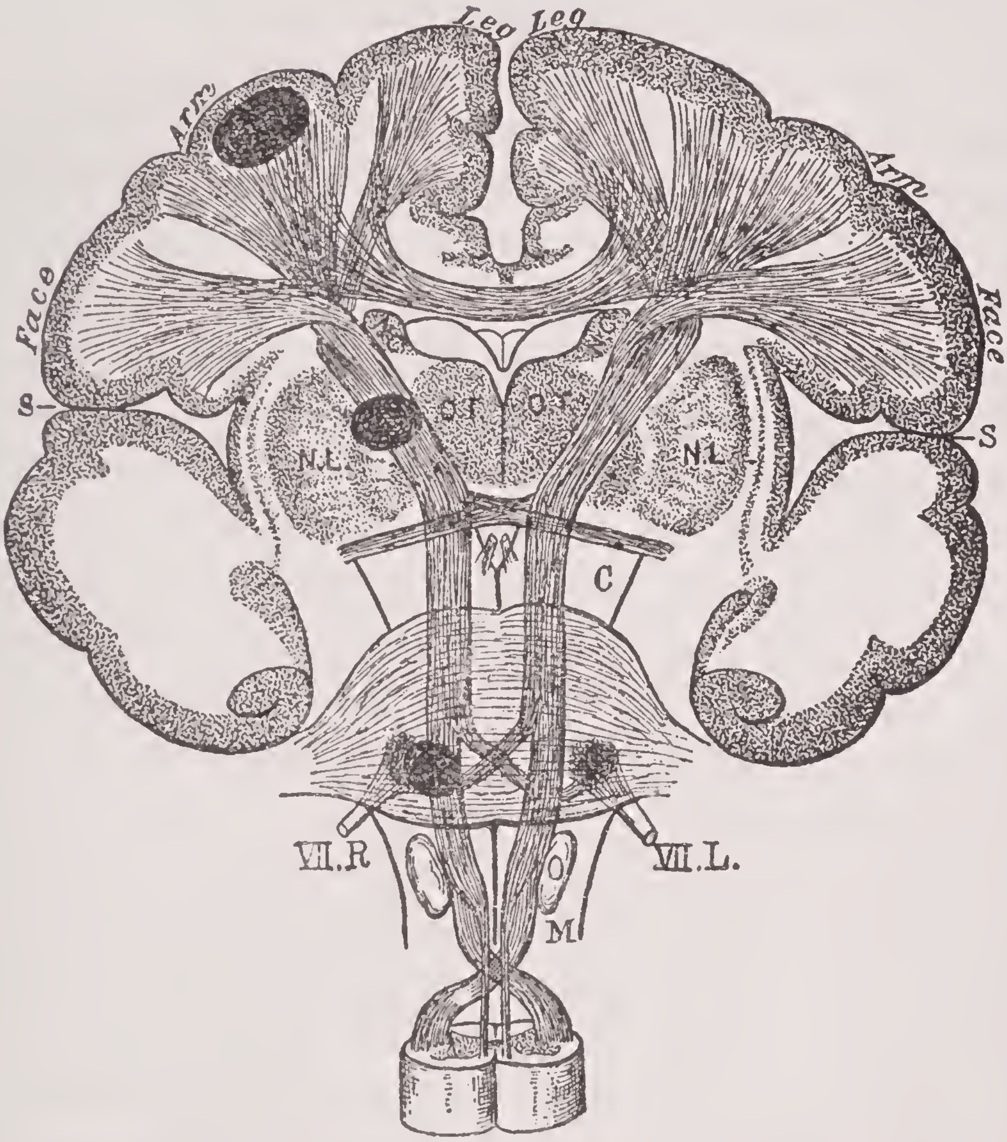


FIG. 51.—The motor tract (Starr): *S*, fissure of Sylvius; *NL*, lenticular nucleus; *OT*, optic thalamus; *NC*, caudate nucleus; *C*, crus; *P*, pons; *M*, medulla; *O*, olivary body. The tracts for face, arms, and legs gather from the lower, middle, and upper thirds of the motor area, pass into the capsule, and through the crus and pons, where the face-fibres cross to the opposite VII. N. nucleus, while the others pass on to the lower medulla, where they partially decussate to enter the lateral column of the cord, the non-decussating fibres passing into the ant. median columns. Lesion in cortex causes monoplegia; in capsule, hemiplegia; in pons, alternating paralysis.

recovery in the majority of cases, and consist of irritability of temper, imperfect memory, bewilderment, delirium, or even dementia. These mental symptoms may clear away or may remain. Crossed facial paralysis occurs with destructive lesions of the lower portion of the pons (see Fig. 51). Small hemorrhages in the cortex cause monoplegia according to their situation. Hemianopsia occurs if the lesion involve the optic tracts or the cuneus.

Prognosis.—Small hemorrhages in the cortex may be recovered from without extensive or permanent paralysis. If the speech-centre be involved, some degree of aphasia may remain. Large central hemorrhages rupturing into the ventricles are rapidly fatal. Hemorrhages into the basic ganglia and the internal capsule may be fatal. In case of recovery from the coma, permanent hemiplegia with contracture is the result. Coma persisting for more than forty-eight hours, congestion and œdema of the lungs, low initial temperature and high secondary rise with delirium and stupor, albuminuria, and the rapid formation of atrophic bed-sores are indications of a speedy termination.

Treatment.—The patient should be kept quiet, with the head high. Ice may be applied to the head, and hot bottles to the feet. In robust patients with high arterial tension venesection should be resorted to; but this is contra-indicated if the blood-tension is low and the pulse is weak.

If the tongue falls back and mucus collects in the throat, the patient should be rolled to one or the other side. Many patients, according to Bowles, are allowed to suffocate from lack of this precautionary procedure. The mouth should be cleansed with antiseptic solutions to diminish the danger of septic broncho-pneumonia. A laxative should be given at the onset—1 or 2 drops of croton oil or $\frac{1}{4}$ grain of elaterium. For throbbing pulse and high tension aconite may be given if venesection cannot be performed.

The paralyzed limbs should be massaged to maintain their nutrition. Faradism is indicated after the lapse of one or two weeks, but when paralysis has lasted for several months and late rigidity with contracture has occurred, further use of electricity is hopeless.

EMBOLISM OF THE CEREBRAL ARTERIES.

Etiology and Synonym.—The usual origin of the embolus is from the valves of the left heart; less frequently the embolus arises from aneurysm or atheroma of the aorta or the great vessels of the neck, or from the lungs. The embolus may be part of a thrombus that has formed in the auricular appendix, most commonly associated with the puerperal state. Septic emboli occurring during the course of ulcerative endocarditis and abscess of the lung give rise to cerebral abscesses, and will be considered under that heading. Embolism is most frequent in young adults, and both sexes are affected in about equal proportions, although, according to the statistics of some authors, the condition seems more prevalent among women. *Synonym:* Cerebral softening.

Pathology.—The middle cerebral artery or one of its branches is occluded in 90 per cent. of all cases, the left artery being more frequently involved than the right. Less frequently are involved the basilar, posterior cerebral, vertebral, anterior cerebral, and the internal carotid. As collateral circulation is never sufficient to maintain the nutrition of the brain-tissue whose blood-supply is suddenly cut off by the occlusion of its nutritive artery, softening and degeneration ultimately result. The nerve-elements are infiltrated with serum and undergo fatty degeneration. If the affected area be the seat of a reflux of venous blood, it will be stained red—"red softening;" later, when the hæmoglobin becomes altered, "yellow softening" results. If there be no reflux of blood, "white softening" results. No matter what the color of the softened spot may be, the actual disease process is the same in all cases. The softening process proceeds rapidly and is usually complete in one or two days. The area of softening may remain unchanged for considerable time, or may be absorbed, leaving a cicatrix which may be pigmented. In other cases the softened area is replaced by a cyst with connective-tissue walls.

The **symptoms** depend upon the artery occluded.

1. *Embolism of the Middle Cerebral Artery.*—(*a*) The *onset* differs from that of hemorrhage in the following particulars: (1) There are no premonitory symptoms; (2) the onset is more sudden; (3) coma is less complete and is shorter in duration; (4) in many cases unconsciousness is not lost, but the patient becomes dizzy and bewildered; (5) convulsive movements of muscles ultimately to be paralyzed occur in one-quarter of the cases; (6) there are no signs of cerebral compression; vomiting, hard pulsating arteries, slow pulse, flushed face, and stertor consequently do not appear; (7) the initial temperature changes are slight, but in a few days fever may develop.

(*b*) *Permanent Symptoms.*—If the trunk of the middle cerebral artery be blocked, hemorrhage and paralysis of the face and tongue occur on the side opposite to the lesion. There may be aphasia if the left cerebral artery is occluded. Attempts at collateral circulation are attended by a decided improvement in the patient's condition in from twelve to thirty-six hours. The improvement may continue or may be but temporary. The sudden onset of hemiplegia, the temporary improvement, and the relapse are characteristic of embolism. The subsequent course resembles that following hemorrhage. The mind in embolism, however, is less frequently affected. If the embolus lodges in a small cortical artery, the softening will be of a small area, so that monoplegia or aphasia alone may develop. In these cases the stage of onset may not be well marked.

The following symptoms follow occlusion of the other arteries :

2. *Vertebral Artery.*—Symptoms of acute bulbar paralysis occur, leading to speedy death.

3. *Basilar Artery.*—There is bilateral paralysis with spasm and rigidity. Symptoms of acute bulbar paralysis occur, and death follows with a high ante-mortem temperature.

4. *Internal Carotid Artery.*—Owing to perfect anastomosis, no symptoms may result. In other cases a transient or permanent hemiplegia may develop.

5. *Anterior Cerebral Artery.*—No symptoms may result, or there may be mental weakness.

Diagnosis from cerebral hemorrhage :

Hemorrhage.	*Embolism.*
Adults between 40 and 80.	Young adults.
Hypertrophied heart.	Endocarditis usual.
Endarteritis.	Not essential.
Right middle cerebral usually.	Left middle cerebral.
Aphasia less often.	Aphasia more often.
Monoplegia rare.	Monoplegia common.
Prodromal symptoms.	No prodromal symptoms.
Coma profound.	Coma slight, transient, or absent.
Convulsions rare.	Convulsions in 25 per cent.
Cerebral compression.	No cerebral compression.

The **prognosis** as regards the attack is, as a rule, somewhat better than in hemorrhage. From the resulting paralysis recovery is not usualy, to be expected.

The **treatment** resembles that of hemorrhage, except that venesection should not be resorted to. Active purgation is not necessary, as in hemorrhage. The heart's action is often so weak and irregular as to require the use of stimulants and digitalis.

THROMBOSIS OF THE CEREBRAL ARTERIES.

Etiology.—Thrombi may form in an artery from disease of its wall, from embolism, or from pressure on the vessel, as from a tumor. Thrombosis may also occur after ligation of the internal carotid artery.

The **pathology** resembles that of embolism, except that the softening occurs more gradually; otherwise the results of the two conditions are identical. The middle cerebral and basilar arteries are those most commonly affected.

The **symptoms** resemble those of embolism, but they appear more gradually. There may be premonitory symptoms—vertigo, transient aphasia or hemiplegia, and drowsiness. Hemiplegia slowly develops, taking several hours for its completion, and the patient gradually becomes comatose.

The **prognosis** and **treatment** are those of embolism.

ANEURYSM OF THE CEREBRAL ARTERIES.

Etiology.—The condition occurs in middle age and is more frequent in men. The etiology is that of endarteritis and aneurysm in general. In many cases aneurysm follows embolism, the embolus disappears, and dilatation follows the secondary inflammatory changes in the coats of the artery.

Pathology.—The aneurysm occurs most frequently on the central, basilar, and internal carotid, less frequently upon any of the branches of the circle of Willis. The aneurysm is usually small, rarely exceeding the size of a cherry, and is usually sacculated.

The **symptoms** are those of tumor at the base of the brain, and hemorrhage. The pressure-symptoms are those of a small basal tumor, the involvement of the cranial nerves being especially frequent. Of suspicious significance is the occurrence of crossed hemiplegia and third-nerve paralysis.

The accompanying illustration (Fig. 52) shows that only pressure on one crus could cause such a distribution of paralysis, and the most likely thing to press on the crus is an aneurysm at the base of the brain.

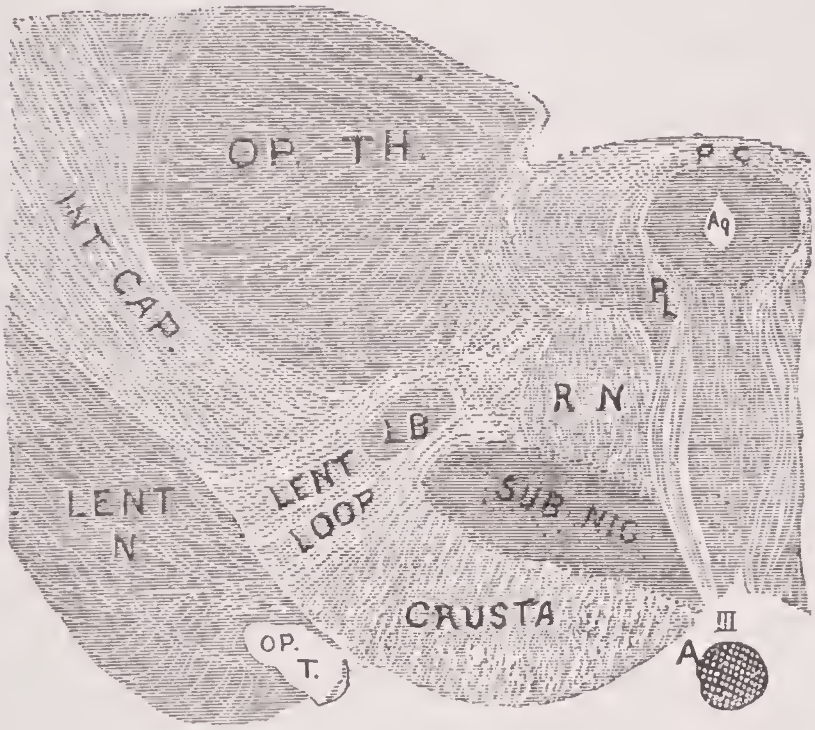


FIG. 52.—Diagram of a section through the crus, etc., in front of the corpora quadrigemina (modified from Wernicke): P C, posterior commissure; Aq., aqueduct of Sylvius; P L, posterior longitudinal fibres; III., third nerve; L B, Luy's body; OP T, optic tract; A, aneurysm causing compression-paralysis of third nerve on same side, and opposite hemiplegia.

The symptoms of rupture lead quickly to a fatal issue, and the sudden occurrence of a large meningeal hemorrhage at the base of the brain is the first information of the disease in the majority of cases.

The **prognosis** is exceedingly bad.

Treatment.—If the diagnosis of aneurysm be made, the patient is to be put to bed, the circulation is to be rendered tranquil, and iodide of potassium is to be given as for aortic aneurysm. The vertebral or the internal carotid artery may be ligated as an extreme measure, but the results of such surgical treatment are not good.

THROMBOSIS OF THE VENOUS SINUSES.

Etiology.—Primary or marantic thrombosis occurs as a terminal event in cachectic conditions, and is not infrequent in the aged. Infants during the first six months of life may be affected, usually after exhausting diarrhœal diseases.

Secondary thrombosis complicates embolism and cerebral tumors producing pressure upon a sinus. In these cases the clot is not septic.

Septic thrombus occurs with disease or injury of the cranial bones or of the middle ear, with meningitis, and with suppurative disease or erysipelas of the scalp, face, or orbit.

Pathology.—The effect of a clot within a venous sinus or a vein is to cause intense congestion and œdema of the brain-territory the circulation of which thus becomes obstructed. Softening of the brain-tissue may ultimately result. Septic thrombi soften, break down, and may give rise to embolic abscesses or to purulent meningitis.

Symptoms.—General cerebral symptoms, which are usually present, consist of apathy, stupor, delirium, convulsions, muscular rigidity, vomiting, optic neuritis, and coma. Localizing symptoms do not usually occur. The cerebral symptoms are most marked when the superior longitudinal sinus is involved, but they are never characteristic. Of diagnostic importance are œdema and distention of the veins outside the skull, in the parts from which the veins pass through the bones to join the internal sinuses, as in the following instances: Thrombosis of the *superior longitudinal sinus* causes congestion and œdema of the sides of the head and forehead, prominence of the anterior fontanelle in children, and epistaxis. Thrombosis of the *cavernous sinus* causes œdema and congestion of the eyelid and a prominence of the eyeball. Thrombosis of the *lateral sinus* causes œdema and congestion over the mastoid.

Septic thrombi give rise to septic symptoms—chills, intermittent or remittent pyrexia, and the "typhoid state." The course of the disease may be complicated by meningitis or abscesses of the brain.

The **duration** of the disease is from a few days to several weeks.

The **prognosis** is bad, except that in case of small, non-septic thrombi recovery may be possible.

Treatment.—The shoulders and the head should be raised in bed to facilitate the venous outflow of blood. The lateral sinus has been explored and septic clots removed, with recovery. Otherwise the treatment is symptomatic.

CEREBRAL ENDARTERITIS.

Etiology.—Cerebral endarteritis usually occurs in males, and is a disease of middle and advanced life. The condition may be due to senile degeneration of the arteries, chronic alcoholism, gout, syphilis, or chronic nephritis.

The **pathology** is that of endarteritis (see Arterio-capillary Fibrosis). The walls of the cerebral arteries are thickened and rigid, and may be the seat of atheroma or of fatty degeneration. Increased connective tissue in the intima may lead to occlusion ("obliterating endarteritis"). Such degenerated arteries cause an irregularly diminished supply of blood to the brain, are subject to spasm, and may lead to softening of the brain, to miliary aneurysms and hemorrhage, or to larger aneurysms of any of the cerebral arteries.

Symptoms.—Three groups of clinical symptoms may be described:

1. *Symptoms of Brain-anæmia.*—The patient suffers from headache, dizziness, spots before the eyes, and buzzing noises in the ears, and becomes unable to concentrate the mind for any length of time. These symptoms are common to brain-anæmia from any cause.

2. *Suddenly-induced Anæmia from Spasm.*—Spasm may occur without known cause at any time, or may follow mental excitement or over-eating or drinking. The spasm is most common in the middle cerebral artery. In mild cases the patient will suddenly develop a partial paralysis of the arm or leg, or will become aphasic. Hemianopia may occur. The onset is usually accompanied by a "wave of faintness" and mental bewilderment, but actual loss of consciousness does not often occur. Spasm may precede the muscular weakness. These attacks are usually temporary, lasting but an hour or so and then wearing off; they are liable to be repeated. In other cases the spasm leads to softening and the symptoms become permanent. In severe cases the patient loses consciousness, although the coma is not as complete as in hemorrhage and is of shorter duration. Hemiplegia with or without aphasia develops, and may be preceded by convulsive movements of the muscles. The temperature rarely undergoes initial changes, and there are no symptoms of compression (slow pulse, stertor, flushed face, and throbbing arteries), as in hemorrhage. The pulse is usually of high tension, owing to the presence of general endarteritis and nephritis. Under favorable circumstances the spasm passes off and recovery takes place in a few hours or days.

Illustrative Case.—Male, sixty-five years; chronic nephritis, general endarteritis. 2 P. M., fell unconscious: right hemiplegia, aphasia; pulse 70, of high tension; no stertor; temperature normal. 12 P. M., conscious; begins to move and to talk. 4 A. M., walked and talked; is rational. 6 A. M., perfectly recovered.

3. *Symptoms of Brain-softening.*—If the spasm be too long continued, or should a thrombus form in the vessel, the brain-tissue, thus deprived of its blood-supply, will die and soften as in embolism or thrombosis. A similar effect is

produced by obliterating endarteritis. In these cases the patient does not recover. If the softened area be extensive, the patient will die in a few days in coma with hemiplegia, death usually being due to pulmonary œdema. In some cases coma is not marked at first, so that paralysis without loss of consciousness is regarded as indicative of acute softening from arterial obstruction, however produced. If a smaller area be involved, the patient will live, but with permanent paralysis or aphasia. In these cases mental derangement is common.

The **diagnosis** is made by the presence of extensive arterial degeneration, by the history of previous attacks followed by recovery, by the absence of symptoms of cerebral compression, by the absence of causes for embolism, and by the rapid recovery under appropriate treatment. When softening occurs the diagnosis from thrombosis cannot be made.

Prognosis.—As the lesion is continuous and progressive, there is liability to recurring attacks which tend to become more and more severe. The prognosis is rendered worse by the nephritis, by the atheroma of the aorta and the coronary arteries, and by the chronic alcoholism if such a habit exists. The danger of hemorrhage must also be considered. The immediate prognosis during an attack depends upon the duration of the spasm and upon the chances of a thrombus forming in the vessel; a positive assurance of recovery, therefore, should never be made.

Treatment.—*Between attacks* the treatment should be directed toward the arterial degeneration (see Arterio-capillary Fibrosis). For the anæmic symptoms the arteries may be relaxed with small doses of potassium iodide, nitroglycerin, or chloral hydrate. Digitalis is contraindicated.

At the time of spasm the arterial dilators above mentioned should be pushed to physiological limits. The bowels should be moved, and the functions of the skin and kidney should be stimulated, to eliminate from the system such noxious products as might cause arterial spasm.

When actual softening occurs treatment becomes inoperative.

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3. DISEASES OF THE BRAIN-SUBSTANCE.

CEREBRAL LOCALIZATION.

Cortical Areas.—1. *The motor area* is located in the cortex of the anterior and posterior central convolutions bordering upon the fissure of Rolando. The area of each hemisphere controls muscular movements of the opposite side of the body. The different groups of muscles are supplied by definite portions of the motor area, as is shown by the accompanying diagram (Fig. 53). Irritation of the motor area gives rise to localized spasm or convulsions ("Jacksonian epilepsy"). Destructive lesions cause paralysis. Slowly spreading lesions, as the growth of a tumor,

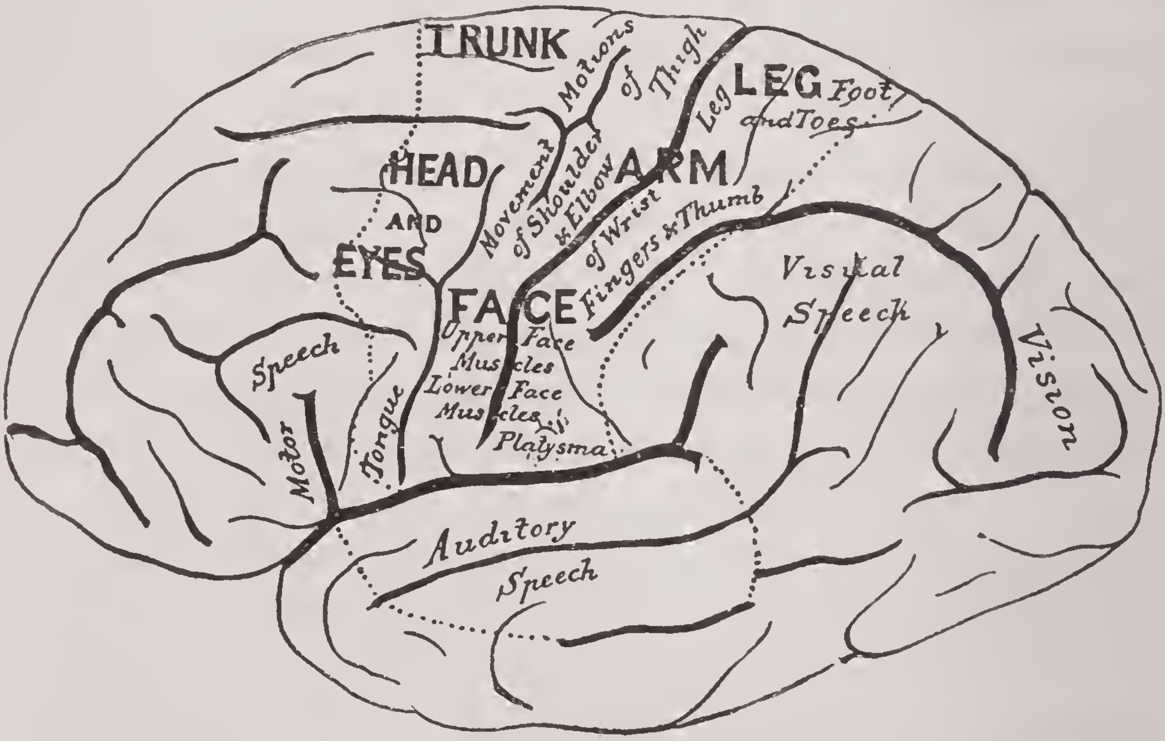

FIG. 53.—The functional areas of the brain, left hemisphere (Starr).

cause spreading irritation followed by destructive symptoms (convulsions followed by paralysis), and involve fresh groups of muscles, so that the size and position of the lesion may be determined accurately. Cortical paralysis is monoplegic and is associated with increased reflexes, but the paralyzed muscles do not atrophy and the electrical reactions are unchanged. Destructive lesions in the motor tract from the cortex to the anterior nerve-cells in the spinal cord are regularly followed by descending degeneration (see Secondary Lateral Sclerosis).

2. The *sensory area* is in the cortex posterior to the motor area, but accuracy in localization cannot be obtained. The tactile sensibility of muscles seems to be in the motor area.

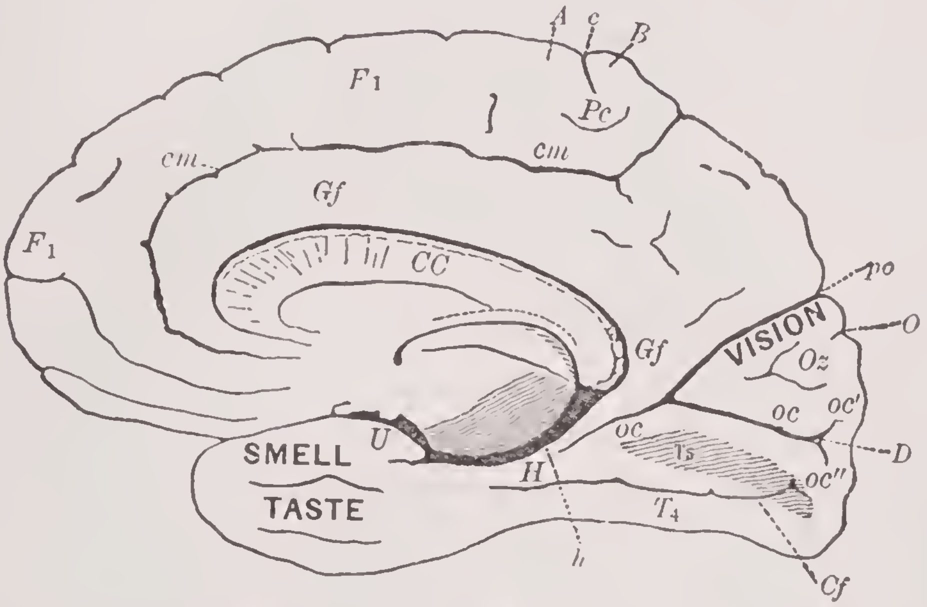

FIG. 54.—Inner surface of right hemisphere: *A*, ascending frontal. *B*, ascending parietal convolution; *c*, terminal portion of the sulcus centralis, or fissure of Rolando; *CC*, corpus callosum, longitudinally divided; *Cf*, collateral or occipito-temporal fissure (Ecker); *cm*, sulcus calloso-marginalis; *D*, gyrus descendens; F_1, median aspect of the first frontal convolution; *Gf*, gyrus fornicatus; *H*, gyrus hippocampi; *h*, sulcus hippocampi, or dentate fissure; *O*, sulcus occipitalis transversus; *oc*, calcarine fissure; *oc'*, superior, *oc''*, inferior ramus of the same; *Oz*, cuneus; *po*, parieto-occipital fissure; P_1'', precuneus; T_4, gyrus occipito-temporalis lateralis (lobulus fusiformis); T_5, gyrus occipito-temporalis medialis (lobulus lingualis); *U*, uncinate gyrus.

3. The *visual area* is in the occipital lobe, including the cuneus on the median surface and the occipital convolutions on the convexity (see Fig. 54). Each area receives impres-

sions from the same side of each retina, so that distinctive lesions cause failure of visual perception in the same side of each retina, the blind field of vision being therefore on the opposite side to the lesion (" homonymous hemianopsia ;" see Fig. 55). Irritation of the visual area causes visual hallucinations. Destruction of the visual area of the left side is followed by word-blindness.

4. The *auditory area* is in the first and second temporal convolutions. Deafness from unilateral lesions is seldom noticeable. Lesions of the auditory centre on the left side are followed by auditory amnesia, or word-deafness.

5. The *smell- and taste-centres* are found at the tip of the temporal lobe, where it rests upon the sphenoid bone. Unilateral lesions do not produce noticeable symptoms.

6. The *speech-centres* are found in the left hemisphere in right-handed people, in the right hemisphere in those who are left-handed. Reflex and automatic speech may receive impulses from both hemispheres. There are four speech-centres :

(*a*) The *motor speech-centre* is in the posterior portion of the third left frontal convolution, and governs the motions concerned in talking. Destructive lesions produce loss of the memory of the effort needed to pronounce a word (" motor aphasia "). Such patients cannot talk or read aloud, but understand what is said to them, and may be able to write.

(*b*) The *auditory speech-centre* is in the first and second left temporal convolutions, and controls the understanding of language and the recollection of the names of things. When the centre is destroyed " word-deafness " results.

(*c*) The *visual speech-centre* is in the cuneus and the occipital convolutions. When destroyed " word-blindness " results, the patient being unable to understand written language.

(*d*) The *writing speech-centre* is supposed to be within

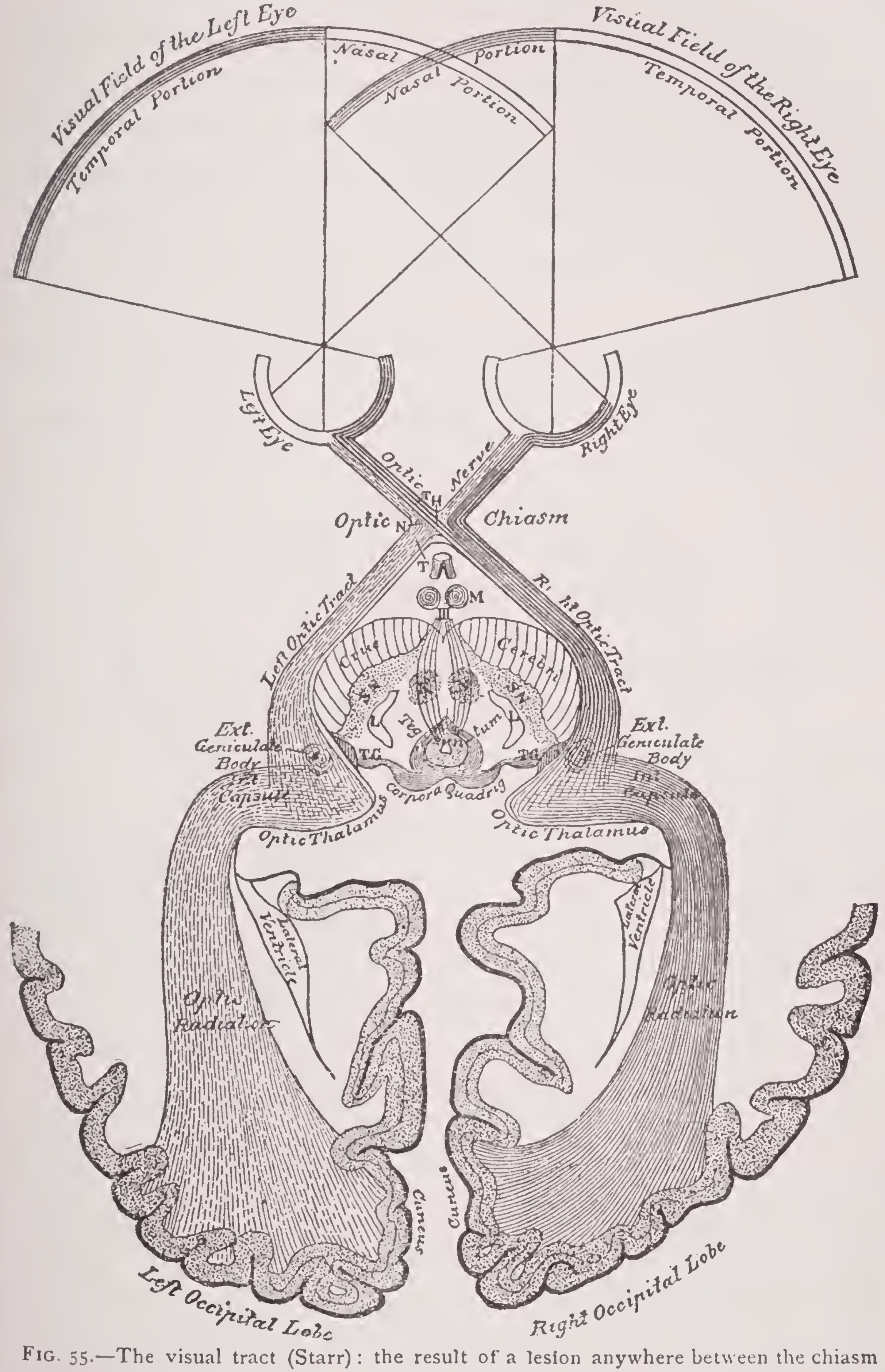

FIG. 55.—The visual tract (Starr): the result of a lesion anywhere between the chiasm and the cuneus is to produce homonymous hemianopsia. *H*, lesion at chiasm causing bilateral temporal hemianopsia; *N*, lesion at chiasm causing unilateral nasal hemianopsia; *T*, lesion at chiasm causing unilateral temporal hemianopsia; *SN*, substantia nigra of crus; *L*, lemniscus in crus; *RN*, red nucleus; *III.*, third nerves.

the motor speech-centre. If the patient be unable to write, the condition is termed "agraphia."

Lesions destroying commissural or association fibres between the various speech-centres cause displacement of words, so that the patient may talk jargon. To this condition the terms "mixed aphasia" and "paraphasia" have been applied.

7. The *intellectual area* is located in the frontal lobes, but no localizing symptoms occur.

Subcortical Localization.—*Centrum Ovale.*—Lesions cause paralysis, which are hemiplegic the nearer the lesion is to the internal capsule, monoplegic the nearer to the cortex.

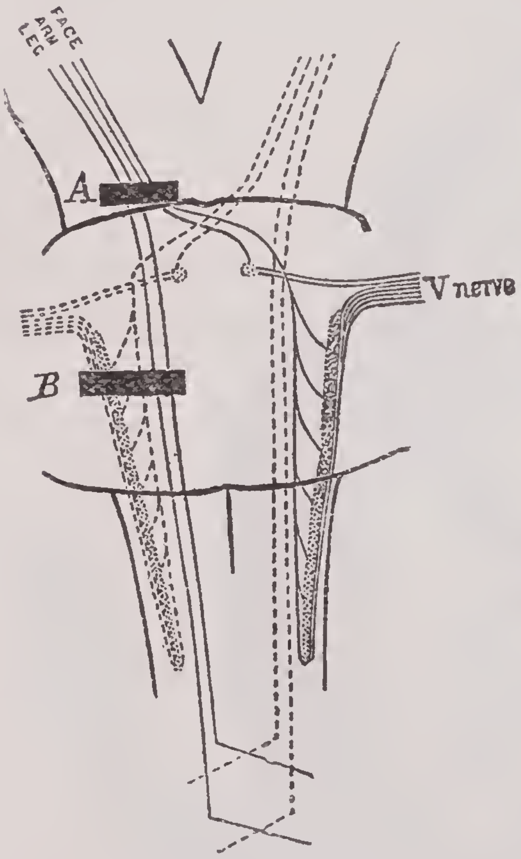

FIG. 56.—The sensory tract in the crus, pons, and medulla, showing nucleus and roots of V. nerve (Starr): *A*, lesion causes hemianæsthesia; *B*, lesion causes alternating hemianæsthesia—left face and right side of body.

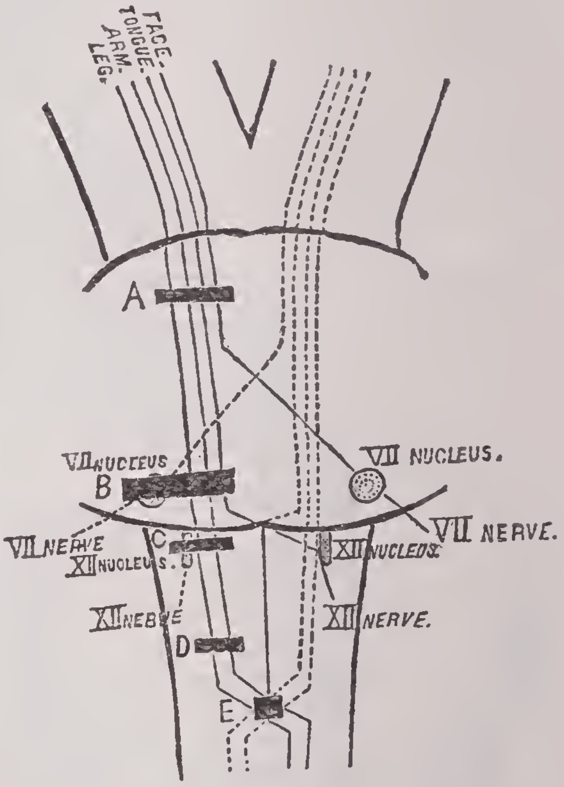

FIG. 57.—The motor tract in the crus, pons, and medulla (Starr): *A*, lesion causes hemiplegia; *B*, lesion causes alternating paralysis—left facial, right extremities; *C*, lesion causes paralysis of limbs and tongue; *D*, lesion causes paralysis of limbs of opposite side; *E*, lesion in motor decussation causes bilateral paralysis.

Hemianæsthesia results from lesions in the posterior portion, and hemianopsia may also result.

Internal Capsule.—Lesions of the anterior two-thirds cause opposite hemiplegia, with involvement of the tongue and face and with descending degenerations (see Fig. 51). The power to wink is maintained. Lesions of the posterior third cause hemianæsthesia.

Lesions of the lower portion of the pons cause crossed facial paralysis (see Fig. 57). The facial paralysis resembles the cerebral form in that the lower fibres alone are involved. If the nucleus of the facial nerve be involved, the patient will not be able to wink, and atrophy and the reaction of degeneration will be developed. Lesions in the lower portion of the pons may also cause alternating hemianæsthesia (see Fig. 56).

Basic Ganglia.—Destructive lesions cannot be diagnosed from those of the tracts which lie near them.

Corpora Quadrigemina.—Destructive lesions cause staggering gait, blindness, nystagmus, and ophthalmoplegia.

Cerebellum.—Lesions cause staggering gait, but the ataxia disappears when the patient lies down. Vertigo is marked. Irritation of the middle peduncle causes staggering toward the side of the lesion; destruction of the middle peduncle causes staggering away from the side of the lesion.

ABSCESS OF THE BRAIN (SUPPURATIVE ENCEPHALITIS).

Etiology.—Infection of the brain-substance may occur through the following channels:

1. *By Continuity through the Cranial Bones.*—Thus abscess arises from injury of the scalp or skull (35 per cent.), from caries of the cranial bones (10 per cent.), from diseases of the middle ear (40 per cent.), or from chronic suppurative

rhinitis (10 per cent.). In these cases the abscess is often associated with meningitis.

2. *Through the Blood-vessels.*—Septic emboli may occur during the course of pyæmia, malignant endocarditis, gangrene, or suppuration of the lung or of the pleura. Abscess of the brain may be a sequel of epidemic influenza.

Abscess of the brain usually occurs between the twentieth and fortieth years, and is more common in men than in women.

Pathology.—Abscess is most common in the temporo-sphenoidal lobe. One-eighth of the cases occur in the cerebellum and are usually secondary to mastoid disease. The abscess at first is limited by jagged and necrotic brain-tissue infiltrated by pus-cells, or, in time, a fibrous capsule may be formed. Rupture into the ventricles may result; rupture through the cortex induces a peracute meningitis. The cerebral sinuses may be the seat of infective thrombi. Embolic abscesses are usually multiple. Abscess of the brain may run an acute or a chronic course.

The **symptoms** of acute cases may be divided into three groups:

1. *Symptoms of Pressure.*—Headache is severe and constant, persists during sleep and stupor, and may be localized. Vomiting is frequent and is not induced by eating. Optic neuritis commonly develops. The pupils are apt to be irregular. The pulse is apt to be slow, running from 60 to 70. Drowsiness alternates with restlessness and delirium, and finally merges into coma.

2. *Toxic symptoms* are those of an internal abscess. A chill occurs at the onset; chills may be repeated at irregular periods throughout the disease. Irregular fever occurs, but usually there are periods during which the temperature is normal or subnormal. The patient finally passes into the typhoid condition.

3. *Localizing symptoms* depend upon the location of the abscess, and whether certain portions of the brain are irritated or destroyed. There may be convulsions (general and epileptiform, or localized in certain groups of muscles), paralyses (either hemiplegic or monoplegic), or aphasia. If phlebitis be present, there will be œdema behind the ear and a sense of fulness over the jugular vein. If meningitis be present, there will be rigidity of the neck and cranial-nerve paralysis.

Abscesses in the frontal lobe may cause only a mental dulness. They are usually caused by disease of the nose or of the ethmoidal cells.

In the temporo-sphenoidal lobe there may be no localizing symptoms. Abscess of the parieto-occipital region may cause hemianopia.

Cerebellar abscess is usually accompanied by vomiting, vertigo, and staggering gait.

A chronic abscess may develop in some portion of the brain that is not highly specialized ("silent region"), become encapsulated, and lead to no particular symptoms. After the lapse of a number of months, however, it is usual for the abscess to show itself by sudden coma or an epileptic seizure. These terminal phenomena are due to rupture either into a ventricle or through the cortex.

The **diagnosis** is based upon the presence of a cause by which infection is possible, upon septic symptoms, and upon the symptoms of a localized destructive lesion.

Prognosis.—Acute abscesses terminate in from three to fourteen days, rarely extending over thirty days. Chronic abscesses may have a latent period of weeks or months, but death usually takes place within one or two days of the rupture. The prognosis is fatal unless the pus can be evacuated.

Treatment is exclusively surgical, and consists of trephining and draining the abscess-cavity. The percentage of cases recovering after such operation is increasing year by year.

TUMORS OF THE BRAIN.

The following are the more frequent varieties of brain-tumors encountered:

1. *Tubercle.*—Tubercular tumors are usually multiple, and are generally found in the cerebellum. They may grow to one or two inches in diameter. One-half of the tumors in children are of tubercular origin.

2. *Glioma* is a form of tumor peculiar to the nervous system, and consists of a fibrillary network and branching cells. A variety called "neuro-glioma" contains large spindle-cells with single nuclei, and others resembling the large ganglion-cells. Gliomata may be hard and may resemble an area of sclerosis, or they may be soft and highly vascular. Glioma is never sharply defined, but fades imperceptibly into the surrounding bone-tissue, hence its invasive tendency. Glioma and sarcoma are often combined.

3. *Syphilitic tumors*, or *gummata*, are most common in the hemispheres and pons, and usually develop from the pia mater or the arteries. Gummata are rarely of large size and are usually multiple.

4. *Sarcoma* usually develops from the membranes or the cranial bones, and may reach large size. Myxo-sarcomata are not uncommon.

4. *Cancer* is almost regularly of secondary growth, and may be multiple. Sarcoma or carcinoma starting from the membranes may invade the bones and appear externally, forming the "fungus hæmatodes," or "perforating tumor of the dura."

6. *Cysts* may be found, resulting from previous hemor-

rhage or from defective development. Hydatid cysts are not uncommon in certain countries, as Iceland and Australia.

Psammoma, *cholesteatoma*, *lipoma*, and *angioma* are rare forms of tumor. An *aneurysm* of one of the cerebral arteries forms a tumor which has elsewhere been discussed.

Pathology.—Except for tubercular and gliomatous tumors, the new growth usually starts in the membranes. According to Starr, the cerebral cortex is involved in 25 per cent., the cerebellum in 25 per cent., the centrum ovale in 5 per cent., the basic ganglia, the pons, the crus, and the corpora quadragemina, each in 10 per cent., other parts in 5 per cent., of all cases. The brain-tissue in the neighborhood of a tumor may be hyperæmic and inflamed, or it may undergo softening. A localized chronic meningitis results if the growth involve the pia mater. If the venous sinuses be compressed, the blood may be dammed back, and serous effusion may take place within the cavity of the ventricles.

Men are more frequently affected than women, in the proportion of 2 : 1. In children, tumors are apt to be tubercular; in early life, gummata or gliomata; in middle life, sarcomata, gummata, or gliomata; in late life, sarcomata, gummata, or carcinomata. Tumors of the brain are rare after the age of sixty.

The **symptoms** of brain-tumors may be divided into two groups: (1) Those due to brain-compression, no matter where the growth may be; (2) the localizing symptoms, due to localized irritation and destruction of a certain portion of the brain.

1. *General symptoms of brain-compression* are tolerably constant in all cases of brain-tumor.

(*a*) *Headache* occurs in 95 per cent. of all cases. It is usually dull and "stupefying," with paroxysmal exacerbations, and it may be general, or localized over the growth, especially in the case of cerebellar tumors. There may be localized tenderness over the scalp. The headache differs from that of functional origin in that the patient gives evidence of pain during sleep or during periods of stupor. The headache of brain-tumor is more severe and persistent than that of any other disease.

(*b*) *Mental disturbances* occur in 85 per cent. of all cases. The patient becomes dull, apathetic, and inattentive. Memory and reasoning are impaired; childishness and even dementia or insanity may develop. The characteristic speech is an articulation in which the syllables are run together. In many cases there are morbid erotic desires.

(*c*) *Optic neuritis* occurs in 80 per cent. of all cases, and is usually bilateral. The old name of "choked disk" given to this condition is inappropriate, as the condition is due, not to pressure, but to a descending neuritis. Double optic neuritis occurs with lead-encephalopathy, meningitis, cerebral abscess, anæmia, and Bright's disease, but an intracranial tumor is by far its most frequent cause. In the first stage the disk is swollen, its edges are blurred and striated, and the physiological cupping disappears. During the first stage there may be no disturbances of vision, but dimness of vision, restricted field of vision, or color-blindness may be noticed. Atrophy of the optic nerve usually supervenes; the disk has a glassy, white appearance, and the arteries are small. In the stages of atrophy impairment of vision, or even total blindness, may result. Optic neuritis, while not a localizing symptom, is most frequent with tumors of the cerebellum and of the basic ganglia.

(*d*) *Vomiting* occurs in 50 per cent. of the cases, and is most marked with cerebellar tumor. The vomiting is "purposeless," has no relation to meals or to local causes, and may assume a projectile character.

(*c*) *Vertigo* is present in 20 per cent. of the cases, and is most constant and severe with tumors of the cerebellum. In many cases vertigo persists while the patient is lying down, so that he will hold on to the side of the bed in order "not to be thrown out." In other cases vertigo is noticed only with a change in position.

(*f*) *General epileptiform convulsions* may occur, and glycosuria and polyuria have been noticed, especially with cerebellar tumors.

(*g*) The *pulse* is usually slower, varying between 50 and 70. The *bowels* are usually constipated.

2. *Localizing Symptoms.*—(*a*) *Tumors of the frontal lobes* give rise to mental dulness, irritability, and dementia. There may be loss of smell. The motor area may finally be encroached upon.

(*b*) *Tumors of the motor area* give rise at first to monospasm ("Jacksonian epilepsy"), preceded by sensory auræ. As the tumor grows the spasms become more diffused, so that by considering the "signal symptom" and the march of the spasm the exact location and rapidity of growth may accurately be determined. Monoplegia succeeds monospasm, the lesion, at first irritative, later becoming destructive. Lesions extending deeply toward the capsule may cause hemispasm and hemiplegia. The paralyzed muscles may, to a moderate degree, be insensitive to pain, touch, and temperature.

(*c*) Tumors of the *parieto-occipital lobe* may give rise to no localizing symptoms. Word-blindness results from lesions of the left angular gyrus.

(*d*) Tumors of the *occipital lobe* produce hemianopia, and, if the left hemisphere be involved, there may result word-blindness and mind-blindness. The latter symptom, also known as "soul-blindness," consists in the incapacity to understand the nature of things which one sees. Bilateral lesions may produce blindness.

(*e*) Tumors of the *temporal lobe* on the left side may cause word-deafness. By extension the motor area may be involved.

(*f*) Tumors of the *basic ganglia* give rise to no characteristic symptoms unless the internal capsule is involved or the lateral ventricles become distended by pressure.

(*g*) Tumors of the *corpora quadrigemina* produce incoordination, forced movements, and oculo-motor paralysis.

(*h*) Tumors of the *crus* cause opposite hemiplegia or hemianæsthesia, with paralysis of the third nerve on the same side as the lesion.

(*i*) Tumors of the *pons* are chiefly productive of paralysis of the nerves emerging from this region on the same side as the lesion, with opposite hemiplegia or hemianæsthesia. Conjugate deviation of the eyes away from the side of the lesion may be observed.

(*j*) Tumors of the *medulla* may cause paralysis of the cranial nerves alone, with the symptoms of bulbar paralysis; or there may be hemiplegia or convulsions.

(*k*) Tumors of the *cerebellum* present distinct peculiarities, especially if the middle lobe be involved. The symptoms consist of marked occipital headache from pressure of the tentorium; marked vertigo, especially if the middle lobe be implicated; cerebellar ataxia, and a pitching or reeling gait. Involvement of the middle lobe causes pitching toward the side of the lesion, forward or backward according to whether the anterior or posterior portion of the lobe be involved. Optic neuritis and vomiting occur early and are marked. The pressure of the growth on the straight sinus causes venous congestion and distention of the ventricles with serum (see Fig. 58). In this way mental slowness and general convulsions may be developed. Pressure of the growth on the medulla may cause bilateral rigidity, increased reflexes, and vaso-motor symptoms in different parts of the body.

The **diagnosis** of brain-tumor may be made from the general symptoms alone. Strict localization is often possible.

The **duration** varies from one to three years. In rare cases the symptoms may persist for a longer time than this.

The **prognosis** is bad, except for gumma and operable growths.

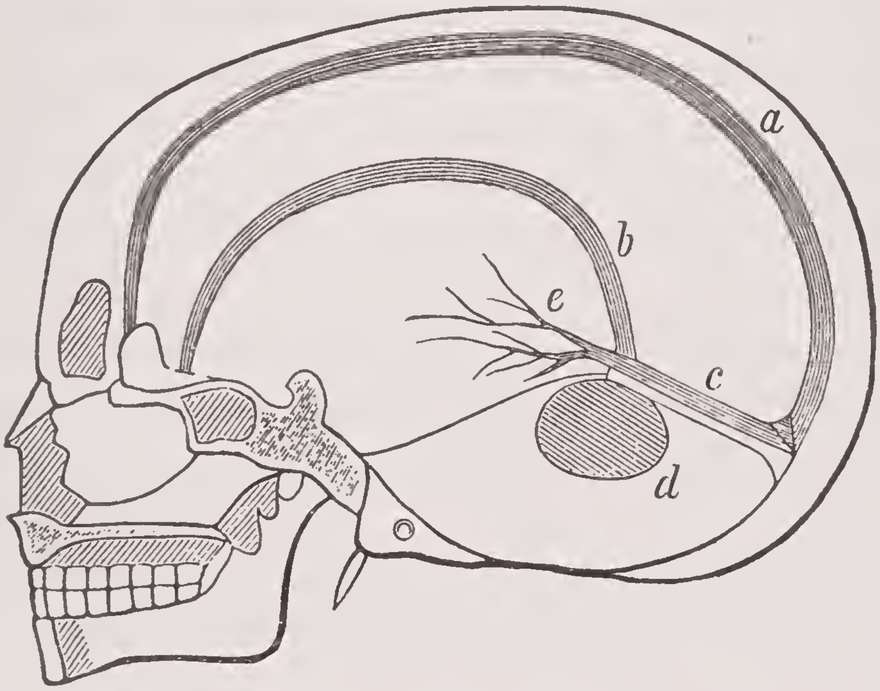

FIG. 58.—Diagram showing the manner in which compression of the veins of Galen is produced by a tumor of the cerebellum, more especially of its middle lobe (after Stephen Mackenzie). The letter *a* points to the superior and *b* to the inferior longitudinal sinus, *c* to the straight sinus, *d* to a tumor beneath the tentorium, and *e* to the veins of Galen.

Treatment.—The patient should be given mercurial inunctions, and potassium iodide up to 300 grains daily. If the growth be gumma, the results are often brilliant. In tubercular tumors a general tonic treatment is indicated, as there are cases reported in which the tubercular tumors have undergone encapsulation and calcific change, and have ceased developing.

The surgical treatment consists in trephining and removing the tumor. Brilliant results have followed this procedure in cases in which the tumor was superficial, accessible, and capable of enucleation. The actual number, however, of operable cases is small (less than 5 per cent., according to Dana). In case of doubt an exploratory trephining is advisable.

CEREBRAL ATROPHY IN CHILDREN.

Etiology.—Cerebral atrophy in children may be congenital, or may be acquired by injuries at birth or by such diseases as occur in adults. In 343 cases collated by Starr, the following primary lesions have been followed by atrophy of the brain in children:

Porencephalus, a localized atrophy in the cerebral hemisphere, which may be deep enough to open into a ventricle —132 cases.

Sclerotic atrophy, atrophy of nerve-elements with increase of connective tissue, involving one or both hemispheres, or part of a hemisphere, or in small scattered areas—97 cases.

Maldevelopment—32 cases.

Atrophy following softening produced by embolism or thrombosis, and limited to arterial districts of the brain—23 cases.

Meningo-encephalitis, shown by thickening and adhesion between the pia and the brain, with atrophy of the cortex—21 cases.

Cysts lying on the brain, causing atrophy by pressure, or associated with atrophy due to the original disease of which the cyst remains as a trace—14 cases.

Hemorrhage on or in the brain—18 cases.

Hydrocephalus with thinning of the brain-tissue—5 cases.

Unilateral hydrocephalus—1 case.

An inflammation of the cells of the motor cortex analogous to anterior poliomyelitis has been described by Strümpell, but lacks positive evidence.

Symptoms.—Four clinical groups may be described:

1. *Infantile Hemiplegia.*—The attacks are usually pre-

ceded by convulsions and fever followed by unconsciousness and hemiplegia. A gradual improvement occurs up to a certain point, after which the symptoms remain permanently. The paralysis may be evident, or there may only be stiffness and clumsiness in performing voluntary motions. There may or may not be aphasia. The paretic muscles show lack of development and are small and cold. Rigidity is due to a condition of exaggerated reflexes. The electrical reactions are unchanged. Essential atrophy of the muscles does not occur. The affected muscles may show tremor or inco-ordinate choreiform movements (" post-hemiplegic chorea "), or there may be involuntary rhythmical spasms of the fingers and the toes (" athetosis "). Various deformities, such as club-foot, arise from the action of the unparalyzed muscular antagonists. Epileptiform attacks occur in one-half the cases. The patient is apt to be imbecile or feeble-minded.

Infantile hemiplegia dating from birth is to be ascribed to meningeal hemorrhage occurring from traumatism during a difficult labor.

2. A double hemiplegia due to meningeal hemorrhage during birth is described as *spastic diplegia* or *birth-palsy*. More rarely the condition follows fevers or convulsions. The extremities are paretic and rigid from over-reflexes. The mental condition is defective, and convulsions are common. The muscles act irregularly (" chorea spastica "), or there may be bilateral athetosis.

3. In some patients mental defects constitute the principal symptoms. In these cases the sclerotic atrophy involves the anterior portion of the brain. The following description is given by Starr:

" The child may be slow in learning to talk ; may seem unable to fix its attention upon anything continuously ; may be exceedingly active, in constant motion—the activity being, however, aimless; may throw things about, or tear things up, or put everything into its mouth; may be very difficult to manage because of its inability to retain and combine impressions with sufficient power to reason upon them; and may therefore be incapable of appreciating the meaning of punishment, if this be inflicted. Such children may have good powers of perception, may recognize persons and objects, show pleasure at bright colors or music or caresses, but fail to show evidence of thought in the sense of reasoning power, judgment, or self-control. Some patients constantly drool at the mouth, cannot be taught cleanly habits, and are manifestly imbecile. Other patients are quite bright in many directions, may even be precocious, show talents in music or drawing, or fondness for mathematics, designing, or languages, yet are apparently unable to appreciate moral ideas, cannot be taught to tell the truth, are cruel and bad, will not control any of their impulses, and so are the distress and despair of parents and teachers. It is those mental qualities which are the product of the highest evolution that have failed to develop in this class of cases. The final result is that they have to be taken care of all their lives, either at home or by attendants, being incapable of supporting themselves or directing their conduct. Many of them have epilepsy."

4. In a fourth set of cases there are neither moral nor motor defects, but the patients are subject to epilepsy, and they present defects of sensory perception. Many cases of deaf-mutism belong to this class. In these patients the atrophy affects the temporal and occipital convolutions.

The **prognosis** is bad for recovery, although life may not be shortened.

Treatment.—Prophylactic treatment consists in cutting short all convulsive seizures in children—by purgatives, hot baths, bromides, chloral, or whiffs of chloroform.

When paralysis occurs, the nutrition of the muscles is to be maintained by massage and passive motion, and deformities are to be corrected by surgical or mechanical measures. For the epileptic attacks bromides may be found useful. In certain cases of arrested brain-development due to microcephalic skull the result of premature union of the sutures, craniectomy may be resorted to. When clots, cysts, or tumors are removable by surgical measures, the chance of recovery is increased. Epileptiform convulsions may in some cases be reduced in severity and in frequency by removing a portion of the skull, so that an increase in the intracranial pressure can occur without producing pressure on the brain.

CEREBRO-SPINAL SCLEROSIS.

Etiology and Synonyms.—The etiology of cerebrospinal sclerosis is obscure. Heredity is marked in a small number of cases. The disease may follow any of the infectious diseases, especially scarlet fever. It is most common in middle life, but it is not infrequently seen in children. *Synonyms:* Insular, Multiple, or Disseminated sclerosis.

Pathology.—Throughout the brain and cord are small grayish nodules of connective tissue which destroy the medullary sheath of the nerves, but leave the axis-cylinder unchanged. The cortex is not usually involved. Sclerosis of the spinal cord alone is of great rarity.

Symptoms.—The onset is insidious and is marked by feebleness and stiffness of the legs and the arms and by increased reflexes. The feebleness may ultimately develop into paralysis, so that the patient becomes bedridden. Marked tremor is developed on motion, but ceases when the muscles are at rest. The speech becomes scanning and "syllabic." Rapid oscillation of the eyeballs (nystagmus) is usually pronounced. The association of volition-tremor with scanning speech and nystagmus forms a characteristic symptom-group. The mental state usually becomes progressively enfeebled. Optic neuritis may develop. Sensation is usually unaffected, but there may be lightning pains and anæsthetic areas as in locomotor ataxia. Vertigo is common. There may be apoplectic seizures, with coma, fever, and transient hemiplegia, or there may be epileptiform convulsions. The patient finally becomes unable to perform the ordinary duties of life, by reason of mental weakness, volitional tremor, and muscular feebleness. The course of the disease is chronic, with periods of temporary improvement.

Diagnosis.—The disease may resemble hysteria or Friedreich's ataxia in children. Anomalous cases are occasionally encountered, which resemble transverse myelitis, locomotor ataxia, or general paresis.

The **prognosis** for recovery is bad.

Treatment is practically unavailing. A course of mercury bichloride with potassium iodide may be tried in alternation with small doses (gr. $\frac{1}{12}$) of nitrate of silver. In all cases a quiet life is to be advised.

GENERAL PARESIS.

Etiology and Synonyms.—The disease is common between the ages of thirty and fifty-five, and is more frequent in men than in women. Heredity appears in 15 per cent. of the cases. The exciting cause may be prolonged mental strain, excesses, syphilis, alcoholism, and lead-poisoning. The disease is not uncommon among active, ambitious business-men. *Synonyms:* Paretic dementia; General paralysis of the insane; Chronic meningo-encephalitis; Chronic periencephalitis.

Pathology.—The membranes are thickened, opaque, and adherent in places to the brain-substance; the cortex is firm and more or less atrophied; there is an increase in the cerebro-spinal fluid. Microscopic examination shows an increase of connective tissue, with a degeneration and disappearance of nerve-fibres and ganglionic cells. In the cord similar sclerotic areas are found in the posterior and lateral columns. The ventricles are usually dilated. There may be small areas of softening in the brain-substance, associated with arterial sclerosis.

The **symptoms** begin insidiously with a change in the moral nature. The patient becomes inattentive and forgetful, and may violate the ordinary rules of decency and deportment. There is increasing mental weakness, with irritability of temper. A peculiar egotism usually but not invariably develops, with delusions of grandeur, so that the patient becomes absurdly boastful, and believes himself to be possessed of millions of money, or to have made the most wonderful inventions, or to be king, emperor, or even God himself. Vaso-motor phenomena and general neurasthenia add their symptoms. In the earlier stages motor symptoms may be noticed: the tongue trembles when it is protruded; the gait is unsteady and shuffling. The pupils are frequently unequal. They may react to distance, but not to light (" Argyll-Robertson pupil "). There may be epileptiform seizures or Jacksonian epilepsy, frequently followed by transient paralyses. The speech becomes thick, owing to the difficulty of pronouncing the lingual and labial consonants. Scanning or a slow, hesitating, monotonous speech is common, words and syllables are frequently omitted, and the patient stumbles over words. Writing becomes uncertain and irregular, and letters or words may be omitted; finally the writing becomes totally illegible. The gait becomes increasingly impaired, and may be spastic or ataxic. The knee-jerk is usually increased. Maniacal outbursts may follow the delusions of grandeur, or there may be periods of melancholia or of depression. In the last stages the patient becomes demented, the bladder and rectum become unretentive, the health fails, and the patient becomes bedridden. Death results from exhaustion or from intercurrent disease.

The course of the disease is progressive, with periods of temporary improvement. The average duration is from three to four years.

The **prognosis** is unfavorable.

Treatment.—In syphilitic cases a thorough course of iodide and mercurials should be employed, but not much is to be expected from the treatment. Nursing and a quiet life in an asylum really constitute the only treatment of the disease.

CHRONIC HYDROCEPHALUS.

An increase in the amount of fluid in the ventricles occurs in a congenital and an acquired form.

Congenital Hydrocephalus.—No known cause has been discovered for this condition. It has occurred in several members of the same family. The lateral ventricles are principally affected, and are distended with fluid, so that the cerebral cortex over them is thin and stretched and may be converted to a thin shell less than a quarter of an inch in thickness. The sutures and fontanelles are widely distended, so that the skull becomes enormously enlarged, in some cases exceeding thirty to thirty-two inches in diameter for a child of two or three years. The bones of the cranium are thinned, the orbital plates are so depressed as to cause exophthalmos. The fluid is limpid, contains traces of albumin and salts, and sometimes contains urea.

Symptoms.—The head may be so large at birth as to interfere with natural labor. In other cases the head does not increase in size until several weeks after birth. The child is restless and irritable. There is difficulty in getting the child to walk, or the legs may be feeble and in a condition of exaggerated reflexes. A few children are bright, but in the great majority some grade of imbecility is present. Strabismus and optic atrophy may develop; nystagmus is commonly present. Vomiting, coma, and convulsions ultimately appear, and the child rarely lives for more than three or four years.

ACQUIRED CHRONIC HYDROCEPHALUS.—This condition may result from compression or obliteration of the straight sinus or of the passage from the third to the fourth ventricle by a tumor; other cases follow meningitis. In a few instances the condition arises without known cause ("serous apoplexy").

The **symptoms** are obscure, and a diagnosis during life is but seldom made. Headache, optic neuritis proceeding to atrophy, and attacks of stupor are commonly observed. The head in the acquired form does not enlarge. There are no localizing symptoms.

Treatment of Hydrocephalus.—Gradual compression of the skull should be made by straps of adhesive plaster crossed in various ways. When pressure-symptoms are present, puncture of the ventricles by a fine aspirating needle and the withdrawal of small quantities of fluid from time to time are justifiable procedures. The subarachnoid sac between the third and fourth lumbar vertebræ may be punctured without risk of injury to the cord, and the fluid slowly removed without much danger of collapse. Medicines are useless, although inunctions of mercury and the administration of potassium iodide have been recommended.

SYPHILIS OF THE BRAIN.

Congenital syphilis of the brain may develop during early childhood, but it is rare. The acquired form is usually a late tertiary manifestation of the disease, although it may develop in from six months to thirty years after the primary sore. The earlier occurrence of symptoms is by some authors attributed to the appearance upon the membranes of an actual syphilitic eruption analogous to the cutaneous exanthems of the secondary period.

1. *Syphilis of the Bones of the Cranium.*—The lesion consists in the formation of spots of dry caries, nodes, and necrosis. Cerebral symptoms arise only if the membranes be secondarily inflamed. If the lesions involve the foramina through which the cranial nerves pass, there will be developed neuralgic pains or motor spasms, followed by anæsthesia or paralysis.

2. *Syphilitic Meningitis.*—The membranes show the lesions of an acute or chronic inflammation, and are invaded by gummata. The symptoms are those of the meningitis and those of the multiple tumors pressing on the cortex, and according to the predominance of either group of symptoms the case will resemble acute or chronic meningitis or cortical tumors. The suggestive points of syphilitic meningitis are—(1) Headache, existing several weeks before the onset of other symptoms, severe in character, and worse at night, preventing sleep; (2) the admixture of symptoms of inflammation of the meninges and of cortical tumors; (3) the rapid improvement under treatment.

3. *Gummata of the Brain.*—The symptoms of brain-tumor are frequently preceded by intense nocturnal headache, by temporary incomplete paralysis of an arm or a leg, or by temporary squint. These partial passing palsies are quite suggestive of cerebral syphilis. The general and localizing symptoms of cerebral gummata have been discussed under the heading of Tumors of the Brain.

4. *Syphilitic Endarteritis.*—The syphilitic changes in the walls of the cerebral arteries were described by Huebner in 1874, and the lesion is known as "Huebner's arteries."

The lesion consists in a thickening of the intima by connective tissue, in some cases leading even to an obliteration of the lumen. Areas of softening may occur in the brain-tissue, from the obliteration of the lumen of the vessel by this new growth or by thrombus-formation. The middle cerebral artery is the one most usually and most extensively affected. The symptoms resemble those of cerebral endarteritis. Syphilitic stupor and paralysis require special description.

Syphilitic Stupor.—The patient complains of severe nocturnal headache, and after a time passes into a peculiar somnolent condition; he may lie for days apparently asleep, or may sit for hours at a time in a torpid, dazed state of mind, answering questions in a peculiar, slow, automatic way, as though talking in his sleep. From time to time the patient may wander about in an aimless fashion. The evidences of severe headache are usually marked, even during the periods of stupor. Prolonged stupor is of serious import, but is not incompatible with complete recovery. The majority of cases, unless relieved by treatment, suddently pass into a condition of profound coma, which is usually fatal.

Syphilitic paralysis comes on suddenly, without loss of consciousness and without exciting cause. The paralysis, which is not complete, and is of a transitory, fugitive character, may be of irregular distribution or may be hemiplegic in type. Oculo-motor paralysis is not uncommon. These fugitive palsies are due to functional brain-disturbance from the diminished blood-supply through the narrower arteries. Should thrombus occur, softening will ensue, so that the paralysis becomes permanent.

5. *Syphilitic disease of the brain* may present nearly the clinical picture of general paresis. The exact pathology of these cases, however, is not known, and it cannot be

asserted as yet that this form of syphilitic brain disease is an independent affection.

The **prognosis** is, upon the whole, favorable, although it should be guarded. More or less recovery is to be expected unless the symptoms indicate an absolute destruction of brain-tissue.

Treatment consist in the vigorous employment of anti-syphilitic drugs. Mercury should at once be ordered by inunction, and pushed until the "gums are touched." Salivation, however, should always be avoided.

Potassium iodide in 30-grain doses three times a day, largely diluted in water or milk, should be pushed rapidly until 300 grains daily are taken, unless the patient show such dangerous symptoms of iodism as hemorrhages. In cases of sudden coma timely venesection may be the means of saving life.

4. DISEASES OF THE SPINAL CORD.

(*a*) Affections of the Meninges.

DISEASES OF THE DURA MATER.

Pachymeningitis externa occurs in an acute and in a chronic form. The acute cases regularly are secondary to inflammation of the vertebral bones or to the extension of neighboring abscesses. The inflammation is fibrino-purulent and gives the symptoms of a compression-myelitis.

Chronic external pachymeningitis is usually due to tubercular disease of the vertebræ. The external layer of the dura is rough, thickened, and covered with cheesy material.

The **symptoms** are due to irritation and compression of the anterior and posterior nerve-roots (hyperæsthesia and motor spasms, anæsthesia, paralysis, atrophy of muscles, and loss of reflexes) and to slow compression of the spinal cord (loss of motion and sensation below the lesion).

Treatment is that of the original disease and that of myelitis.

PACHYMENINGITIS INTERNA HÆMORRHAGICA (Hæmatoma of the Dura Mater).—This disease is usually associated with a similar affection of the dura mater of the brain, and the two lesions are identical, so that a further description of the disease process is not necessary. The cervical region is the one usually affected. The symptoms are those of chronic spinal meningitis—pain in the back, motor and sensory irritation and impairment. Hemorrhages occur from time to time, causing sudden exacerbations of the spinal symptoms and compression-symptoms (see Meningeal Hemorrhage).

Treatment is unavailing.

PACHYMENINGITIS INTERNA HYPERTROPHICA.—The dura becomes thickened by fibrous tissue, which irritates and destroys the nerve-roots and causes slow compression of the cord. The thickening of the dura generally extends above the cord like a ring, and is usually limited to the cervical region.

Symptoms.—1. *Stage of Irritation.*—The symptoms are due to irritation of the anterior and posterior nerve-roots. Neuralgic pains develop in the course of the affected nerves, and are referred to the neck, arms, and the upper portion of the thorax. There are areas of hyperæsthesia with tingling sensations. Motor symptoms consist of spasm and rigidity of the neck and of the muscles of the upper extremities.

2. *Stage of Destruction.*—Hyperæsthesia and neuralgia give way to anæsthesia. Paralysis with atrophy and loss of reflex succeeds the muscular spasms. Deformities result from the atrophy and paralysis. If the lower cervical enlargement be compressed, the ulnar and median nerves are

chiefly affected, so that over-extension of the hand results. If the lesion be higher up, the musculo-spiral nerve is affected, so that the hand will drop. Secondary degeneration of the pyramidal tract results from the pressure-myelitis, and spastic paraplegia develops (see Secondary Lateral Sclerosis).

The **prognosis** is bad for recovery, although death usually results from intercurrent disease. In some cases the disease may be arrested, and the patient live for years with permanent contractures and deformities.

Treatment consists of counter-irritation to the affected portion of the spine, and the empirical use of potassium iodide.

DISEASES OF THE PIA MATER.

Acute Leptomeningitis.

Etiology.—Acute inflammation of the pia covering the cord may be due to extension of a similar inflammation of the pia of the brain, being thus part of the lesion of a simple or an epidemic meningitis; or it may be due to traumatism of the vertebræ, or to operation, such as the opening of a spina bifida. The disease may complicate certain acute infectious diseases, especially pneumonia, small-pox, scarlet fever, and typhoid fever. A tubercular inflammation of the spinal meninges may also occur. In a few cases the exciting cause seems to be exposure to wet and cold. The infecting germ may be that of epidemic cerebro-spinal meningitis (*q. v.*), the cocci of pus, the pneumococcus, or Eberth's bacillus.

Pathology.—The lesions may be diffused throughout the length of the cord or may be localized in the cervical region. The pia is congested, thickened, and infiltrated with fibrin, serum, and pus. The exudation is usually more abundant in the posterior portions of the pia, owing to gravitation when the patient lies upon his back. The peripheral portions of the cord are usually infiltrated by inflammatory products. The nerve-roots may also be involved. In the majority of cases similar lesions are found in the cerebral pia mater.

The **symptoms** are due in the first stage to intense irritation of the spinal nerves. Pain in the back and shooting pains along the nerves, with areas of hyperæsthesia, are the prominent sensory symptoms. Irritation of the motor nerves gives rise to spasm and rigidity of the affected muscles. The spine is stiff and rigid; the head is thrown back; there may be opisthotonos. Owing to the retraction of the head and neck, the larynx may be so firmly pressed against the spinal column as to cause obstructive dyspnœa with stridor. The reflexes are generally exaggerated. There may be retention of urine from reflex spasm of the bladder. During the earlier stages there is no paralysis, though the muscles may be kept quiet, so as not to increase the neuralgic pains. Fever is regularly present, but runs an atypical course, rarely exceeding 104° F. Finally the second stage develops, in which hyperæsthesia is succeeded by anæsthesia, the pains cease, and complete paralysis supervenes. There may now be developed bed-sores and paralysis of the sphincters. Reflex irritability becomes lost.

The **diagnosis** may be difficult. Symptoms of spinal meningitis may be present in cases in which the meninges are afterward demonstrated to be normal, while well-marked cases of leptomeningitis, especially of the cerebro-spinal form, may be unattended by characteristic symptoms. The diagnosis from tetanus is made by the absence of trismus and of the risus sardonicus and by the intensity of the pains. Myelitis is to be excluded by the marked and continued hyperæsthesia, by the late appearance of paralysis, and by the bladder not being involved.

The **prognosis** is not good, although the acute condition may subside and symptoms of chronic meningitis develop. Dyspnœa the result of spasm or paralysis of the respiratory muscles is a most unfavorable symptom.

The **treatment** is practically that of acute myelitis.

CHRONIC LEPTOMENINGITIS.

Etiology.—Chronic spinal meningitis may follow an acute attack or may be chronic from the start. In the latter case the disease may be due to syphilis or alcoholism, or the lesion may complicate chronic diseases of the cord that extend so as to involve the membranes, or extra-medullary lesions, such as tumors or disease of the vertebral bones.

Pathology.—When the condition follows an acute attack, the lesions usually involve an extensive area of the membranes; in other cases, chronic from the start, the distribution is more limited. The pia is thickened and adherent to the cord and the dura. The nerve-roots may be compressed, and may even become atrophied. The cord usually shows increase of connective tissue in the cortical zones.

The **symptoms** resemble in kind those of the acute form, and are due to the involvement of the nerve-roots, but muscular spasms are much less prominent. There is pain in the back, radiating along the course of the nerves, with areas of hyperæsthesia. Stiffness of the back and generally increased reflexes are present in the earlier stages. The rectum and the bladder are but seldom involved. Motor weakness gradually merges into paralysis, with atrophy of the muscles and loss of reflexes. These symptoms result from atrophy of the anterior nerve-roots through compression by the thickened pia. The symptoms of meningitis are frequently accompanied by those due to a primary disease in or outside of the spinal cord.

Prognosis.—The disease is chronic in its course, extending over years. Recovery cannot be expected.

Treatment is that of the chronic stage of myelitis.

HEMORRHAGE INTO THE SPINAL MEMBRANES.

Etiology and Synonyms.—This rare condition may result from (1) blows and concussions, (2) chronic pachymeningitis hæmorrhagica, (3) rupture of an aortic aneurysm into the spinal column after erosion of the vertebral bones, (4) hemorrhagic diseases, as purpura hæmorrhagica, and scurvy, (5) after convulsions or tetanus, (6) or as a lesion of caisson-disease. Blood effused into the cranium, as in hemorrhages at the base, may make its way down between the membranes of the cord. This latter diffusion of blood occurs most commonly with rupture of an aneurysm of the vertebral or basilar artery. *Synonyms:* Extra-medullary hemorrhage; Hæmatorrhachis; Spinal apoplexy.

Pathology.—The blood may be found between the dura and the bones (extra-meningeal hemorrhage), and is extravasated from the large plexus of veins lying outside the dura. In other cases the blood is found between the membranes (intra-meningeal hemorrhage). Hemorrhage is most common in the cervical region, but it may occur anywhere. In extensive extravasations the spinal cord may be compressed, but the amount of blood is rarely sufficient for this.

Symptoms.—The characteristic features of hemorrhage within the spinal membranes are the sudden appearance of symptoms of an intense meningeal irritation without initial fever. Pain develops suddenly in the back and radiates along the course of the nerves. There are muscular rigidity and spasm. Motor and sensory impairment may ultimately appear, but seldom to a great extent. If the hemorrhage be in the cervical region, the pain is felt in the arms, the neck is rigid and immobile, and dysphagia, interference with respiration, and dilatation of the pupils may appear. Larger hemorrhages in the upper cervical region may be immediately fatal. Occurring in the dorsal region, the pain encircles the abdomen. Hemorrhage in the lumbar region causes pain in the legs; there may be spasmodic retention of urine. If the hemorrhage compress the cord, compression-myelitis will result, with paraplegia and anæsthesia, and the rectum and bladder will be involved.

The **prognosis** is bad, but not hopeless. Perfect recovery may follow slight extravasations.

Treatment.—The patient should lie on the face while ice-bags are applied to the spine. Ergot may be given at the onset in full doses. After the hemorrhage has ceased the treatment is that of myelitis.

(*b*) Diseases of the Blood-vessels.

ANÆMIA.

The etiology and symptomatology of spinal anæmia are but little known. In profound anæmia there may be no symptoms referable to the cord. It is known that ligature of the abdominal aorta in animals is followed by paraplegia, and in man this paralytic condition has followed sudden blockage of an aneurysm of the abdominal aorta by a thrombus or by a detached portion of the clot. A transient paraplegia has also followed sudden and profuse hemorrhage or exhausting diarrhœa. The weakness of the legs in those suffering from aortic regurgitation is supposed to be due to spinal anæmia.

HYPERÆMIA.

Acute hyperæmia results from sexual excesses, physical over-exertion, sudden cessation of the menses, and from over-dosing by strychnine.

The **symptoms** are indefinite. There may be numbness, with neuralgic pains, muscular twitching, and weakness, these being usually confined to the lower extremities.

Treatment consists in the application of an ice-bag or of cups to the spine. The patient should lie on the side or the face. Sodium bromide may be given to control the symptoms. Ergot, although recommended by some, does not seem to be of as much service as is generally supposed.

Chronic hyperæmia may complicate various lesions of the cord and membranes, but the symptomatology is obscure.

HEMORRHAGE INTO THE CORD.

Etiology and Synonyms.—Hemorrhage into the cord has been found with tetanus, strychnine-poisoning, and conditions leading to sudden asphyxia. Hemorrhage commonly complicates inflammations and tumors of the cord. Primary hemorrhage, which may be due to blows or falls, to convulsions, or to hemorrhagic conditions, is most common in young males. *Synonyms:* Hæmatomyelia; Intramedullary hemorrhage; Spinal apoplexy.

Pathology.—The clot is rarely larger than an almond, and is usually situated in the central gray matter. The nerve-structures are lacerated at the seat of hemorrhage, resulting in a total transverse destructive lesion of the cord.

The **symptoms** resemble those of acute transverse myelitis, but there is no initial fever, the paraplegia is suddenly induced, and the stage of irritation is not marked. The case may resemble one of spinal meningeal hemorrhage, but in the latter condition pains and spasm are more marked than anæsthesia and paraplegia, and there is more liability of recovery.

The ultimate course of spinal hemorrhage is that of a chronic transverse myelitis, and in many cases it is impossible to say whether the case is one of primary hemorrhage with myelitis or of myelitis with secondary hemorrhage.

The **prognosis** is not good. Many cases die during the acute symptoms, while others live for years with permanent paraplegia. In these cases the prognosis is that of chronic myelitis.

The **treatment** is that of myelitis.

Embolism and thrombosis are rare conditions. Embolism may be suspected, should a patient with valvular disease suddenly develop paraplegia and paranæsthesia without other apparent cause.

CAISSON-DISEASE; DIVER'S PARALYSIS.

Etiology.—Persons who work in caissons and diving-bells under an increased atmospheric pressure may develop this disease when they suddenly emerge into the normal atmosphere. The pressure must be more than three atmospheres, and the longer they work in the caisson and the more suddenly they return to the surface, the greater is the liability to the disease. Those unused to the work are most frequently attacked.

The **pathology** is somewhat obscure. Under raised atmospheric pressure the blood is driven from the surface to the internal organs. When the pressure is reduced to the normal, vascular disturbances are supposed to result in the cord, with spots of congestion and small hemorrhages which may ultimately result in myelitis. Another theory is that an excess of nitrogen is absorbed by the blood when under high pressure, and that when the pressure is too suddenly reduced bubbles of this gas are liberated and lacerate the nerve-structures of the cord.

The **symptoms** may appear at once or may be deferred half an hour or more after leaving the caisson. In most cases there are agonizing neuralgic pains in the limbs, the legs are tender to the touch, and there is some loss of motor power. There are apt to be epigastric pain, nausea, vomiting, headache, and dizziness. In severe cases paralysis and anæsthesia rapidly develop in the legs, although neuralgic pains may still continue. There may be temporary loss of consciousness.

Prognosis.—The patient may convalesce in a few days, or recovery may result only after weeks or months of suffering and paralysis. Severe cases may pass into coma, and die in a few hours or days after the onset. Atrophic bed-sores and cystitis may complicate the course of the protracted cases. In some instances the patient is left with permanent paraplegia and the symptoms of transverse myelitis.

Treatment.—A sufficient time must be spent in passing through the lock, in which the pressure is reduced. At least five minutes should be spent for each atmosphere. Workmen should be carefully selected and gradually accustomed to the work, and the hours of labor in the caisson should at first be short. When the symptoms occur, it is recommended that the patient should at once be put back under a slight atmospheric pressure. The use of ergot in large doses seems to be of service during the acute stages of the disease. Morphine may be required for the neuralgic pains. The paralytic symptoms are to be treated on the same principles as those governing the treatment of myelitis.

(*c*) Diseases of the Substance of the Cord.

ACUTE ANTERIOR POLIOMYELITIS (ATROPHIC SPINAL PARALYSIS).

Acute anterior poliomyelitis occurs both in children and in adults. An infantile and an adult form are to be described.

ANTERIOR POLIOMYELITIS IN CHILDREN.

Etiology and Synonym.—The disease occurs in children between two and five years of age, and is equally frequent in boys and girls, in weakly and in robust subjects. The majority of cases appear during the warm summer months. There may be a history of exposure to the hot sun, of a fall or an injury, or the condition may follow some acute infectious disease, especially measles. In some instances it would seem that the disease itself was of an infectious origin, and epidemics of the disease have even been recorded. *Synonym:* Infantile paralysis.

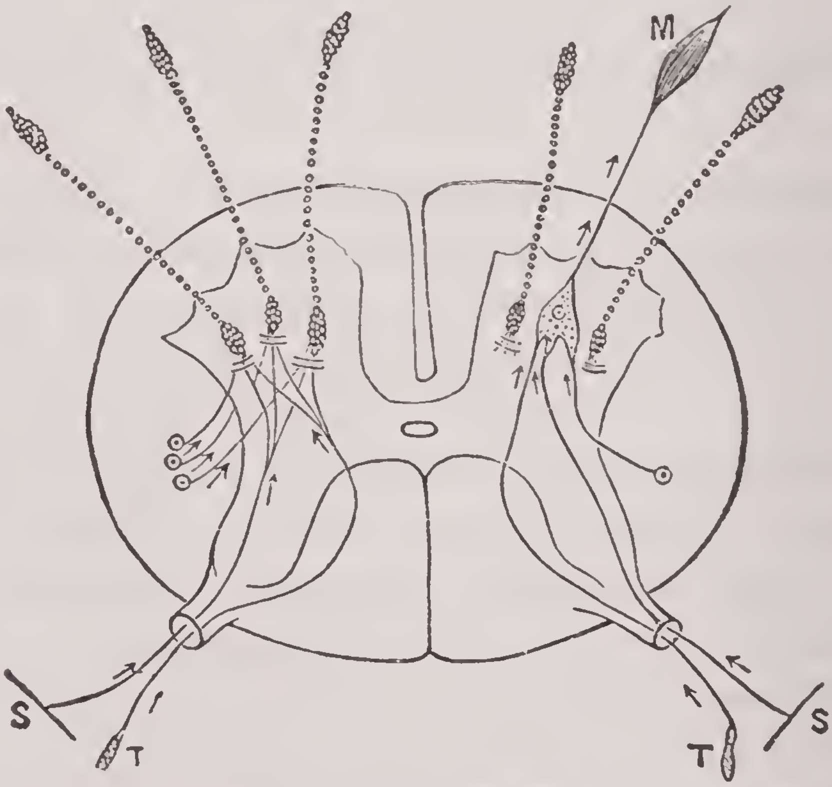

FIG. 59.—Diagrammatic representation of the symptoms that result from acute destruction of the anterior cornua of the spinal cord (Bramwell). On the left side the destruction of the nerve-cells is complete: the anterior nerve-roots, motor nerve-fibres, and the muscles which they supply are all degenerated; there is a total "block" to the passage of voluntary motor and reflex motor impulses. On the right side two-thirds of the motor cells are destroyed; two-thirds of the muscular area connected with the right anterior cornu are degenerated and atrophied; one-third (M) remains healthy, and can be made to contract by voluntary or reflex motor impulses.

Pathology.—The lesion consists in the degeneration of the anterior motor cells, preferably of the lumbar and cervical enlargements. The ganglionic cells become swollen and degenerated, and may either return again to a normal condition or may become atrophied.

During the earlier stages the gray matter about the motor cells becomes congested and infiltrated by leucocytes; later the inflammation becomes chronic and results in an increased production of connective tissue. As the anterior motor cells preside over the nutrition of the anterior nerves and the muscles to which they are distributed, granular degeneration and atrophy of nerve and of muscle follow the destructive lesion of the motor cells. The lesion is usually bilateral, and affects groups of cells functionally associated (Remak describes an " upper-arm type," in which the supinator longus is paralyzed, with the biceps and the brachialis anticus). Through the anterior motor cells pass fibres from the central regions of the cord, controlling vascularity and general nutrition, so that destructive lesions of the anterior cells are regularly followed by imperfect growth, cyanosis, and a lowered temperature of the affected members.

In cases of long standing the affected cornua become small and atrophied, and slight sclerotic changes are found in the pyramidal tract.

The **symptoms** may be divided into three stages:

1. *The Stage of Onset.*—The onset begins abruptly, with fever of from 100° to 103° F., usually accompanied by convulsions, twitching of the muscles, delirium, or even coma. There may be, in older children, some complaint of aching in the joints. The symptoms may be well marked, or there may be only moderate fever which may pass unnoticed. The symptoms of this stage last for a few hours or for several days.

2. *The stage of paralysis* is distinctive in that the greatest degree of paralysis is reached at the onset, and any change

afterward is for the better. When the paralysis has remained stationary for twenty-four hours the danger of further extension is extremely small. The distribution of the paralysis depends upon the situation and extent of the lesion. The type is paraplegic, and the legs are more frequently affected than the arms, in the proportion of 4 : 1. One or both legs may be paralyzed, or the muscles of the upper extremities may be affected as well, or may be paralyzed alone, or one arm and one leg may be involved. The paralyzed muscles are those functionally associated. The extensors are more frequently involved than the flexors. All the muscles of a limb are but rarely affected. After a stationary period of from two to six weeks some of the paralyzed muscles acquire their former power, while others remain paralyzed. The affected muscles are flabby, undergo wasting, and there is absence of all reflex action. Fibrillary twitchings may occur in the paralyzed parts, and the skin is bluish and cold to the touch. The affected muscles show the reaction of degeneration,[1] there being at first an

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[1] *Reaction of Degeneration* (R. D.).—Since contractions only occur on closing or opening the galvanic current, and as there are but two poles, the *anode* or positive and the *cathode* or negative, there are of necessity but four possible forms of contraction:

1. When the cathode is on the muscle and the anode upon a neutral and distant point—(*a*) the contraction which occurs on closing the current (the cathodal closure contraction, or C. C. C.); (*b*) the contraction which occurs on opening the current (cathodal opening contraction, or C. O. C.).

2. When the anode is on the muscle and the cathode upon a distant neutral point—(*a*) the contraction which occurs on closing the current (anodal closure contraction, or A. C. C.); (*b*) the contraction which occurs on opening the current (anodal opening contraction, or A. O. C.).

These four forms of contraction occur in a definite order of intensity, which order differs in health and in disease. In health the order is—C. C. C.–A. C. C.–A. O. C.–C. O. C.

The A. O. C. and the C. O. C. are usually evident only with currents of such intensity as to cause pain.

In degeneration of the anterior nerve or of the motor cells of the anterior increase and then a decrease in the galvanic irritability, and in two weeks or sooner there is a total loss of faradic excitability.

Negative symptoms are important: (1) There is no secondary involvement; (2) the bladder and the rectum are unimpaired; (3) trophic changes in the skin do not occur; (4) there is no change in the mental condition nor in the general health; (5) the cranial nerves are not involved.

3. *Symptoms of permanent deformity* are due (1) to the condition of the paralyzed limb—the growth is retarded, the limb appears atrophied, withered, cold, and bluish; (2) to increased mobility of the joints, owing to the relaxed condition of the paralyzed muscles; (3) to muscular contracture of the unparalyzed muscles, the type of which deformity is club-foot.

The **prognosis** for life is good, but perfect recovery from paralysis is not to be expected, although a certain amount of improvement almost always occurs. The following rules may be given in the way of estimating the extent of permanent paralysis: Muscles which in two weeks respond to faradism will surely recover, while muscles not thus responding will remain paralyzed for a greater or less time. Muscles which in three months show a return of irritability to faradism will recover partially. Muscles which do not respond to faradism in six months will never recover.

It is important never to give too gloomy a prognosis as long as the faintest contraction is produced by the faradic current.

Treatment.—During the stage of onset the spine should be cupped and fever be controlled by the ordinary measures. A brisk laxative should be given at the onset. During the

cornua the order is changed to the "reaction of degeneration," in the following order of intensity: A. C. C.–C. C. C.–C. O. C.–A. O. C. The character of the contraction is also changed, being slow, prolonged, and even tetanic.

earlier part of the stage of paralysis ergot and sodium bromide with small doses of potassium iodide should be given until some amount of improvement appears. During this time the paralyzed members should be wrapped in cotton. In two or three weeks, when the acute stage has passed, strychnine should be administered, as in the following prescription:

℞.	Strychninæ sulphat.,	gr. $\frac{1}{60}$;
	Ferri pyrophosphat.,	gr. j;
	Acid. phosphoric. dil.,	♏ iv;
	Syrup. zingiberis,	ad ʒj.—M.

Sig. Such a dose three times a day to a child from three to five years of age.

The affected parts should be carefully and persistently rubbed morning and evening, and the muscles should at the same time be gently kneaded. The faradic current should be applied daily to such muscles as respond, while to the paralyzed muscles the slowly interrupted galvanic current should be applied.

For the permanent deformities much good can be done by various apparatus or by surgical measures.

Anterior Poliomyelitis in Adults.

Males are more usually affected than females. The etiology and pathology are the same as in children, but the following clinical differences are observed: (1) The onset is less acute; (2) the cranial nerves may be involved in some cases; (3) rheumatoid pains may be present, and are referred to the affected muscles and joints; (4) muscular tenderness may be extreme; (5) fewer muscles are affected than in children, and recovery is usually more complete; (6) owing to the development of the limbs of an adult, the withering and retarded growth of the affected member are not so noticeable.

Although the adult cases of anterior poliomyelitis are described as the counterpart of the same disease in children, there is strong probability that the disease is really a multiple neuritis. Further observations are necessary to determine this point.

SUBACUTE AND CHRONIC POLIOMYELITIS.

Synonym.—Duchenne's paralysis.

It is undecided whether this disease is a chronic poliomyelitis or a multiple neuritis. The pathology is therefore obscure.

Etiology.—The disease is rare, and is almost exclusively confined to adults.

Symptoms.—The onset is gradual, and is characterized by motor weakness which increases in extent and ultimately leads to paralysis. The paralyzed muscles rapidly atrophy, show absence of reflexes, and the R. D. can be obtained. There is no sensory involvement, nor is the bladder or the rectum implicated. The symptoms usually begin in the legs and extend to the trunk and the upper extremities (ascending type); or the disease may be first noticed in the arms, and may spread to the legs (descending type). The early stages of the descending type, in which the arms are first affected, may closely resemble lead-paralysis.

Prognosis.—In the majority of cases a certain degree of improvement is noticed in a few weeks or months, but recovery is incomplete except in rare cases. In some instances death results from an extension of the disease to the medulla, with the symptoms of bulbar paralysis. The duration of the disease is usually from one to four years.

Treatment is that of the acute poliomyelitis after the febrile stage has passed.

PROGRESSIVE MUSCULAR ATROPHY.

Etiology and Synonyms.—The disease is most frequent in males between twenty-five and forty-five years of age. An hereditary influence is often observed, and there are cases in which the disease has been handed down for five generations. It is possible that some of these hereditary cases, however, are really examples of idiopathic muscular atrophy. Excessive physical exertion, mental worry, exposure to wet and cold, syphilis, and chronic lead-poisoning have been ascribed as exciting causes; the affection has been known to follow measles, typhoid fever, and acute rheumatism. *Synonyms:* Wasting palsy; Chronic anterior poliomyelitis.

Pathology.—The lesions are found in the muscles, nerves, and cord. The muscles are wasted and pale, and the fibres are shrunken. Certain fibres are seen to have entirely disappeared, leaving empty and collapsed sheaths. The wasting of the muscle is not *en masse*, but fibre after fibre is picked out in the process of atrophy. There may be an increase in the interstitial tissue. The atrophy of the fibres is simple, and granular degeneration is not apparent as in infantile paralysis. The anterior nerve-roots emerging from the affected part of the cord undergo simple atrophy, and fibre after fibre disappears. In the cord the lesion is practically confined to the anterior cornua. The motor ganglionic cells waste, lose their processes, and one after another undergoes simple atrophy (Fig. 60). Sclerotic changes are usually found in the direct and crossed pyramidal tract, and when these changes are sufficiently marked to give rise to symptoms, the name of "amyotrophic lateral sclerosis" is given to the resulting complex lesion. This form of disease will be separately considered. In many cases the disease extends upward to involve the motor nuclear cells in the medulla.

The **symptoms** begin gradually and insidiously. It is noticed that the muscles tire easily, are not as firm as normal, and ache after slight exertions. Fibrillary contractions may be observed. The atrophy is usually first noticed in the interossei muscles and in those of the ball of the thumb. Motor weakness is proportionate to the extent of the atrophy, as there is in this disease no essential paralysis. Advanced atrophy of the interossei give the hand the "griffin-claw" appearance, in which flexion of the last two phalanges is associated with extension of the first phalanx at the metacarpal joint. Occasionally the deltoid is the first

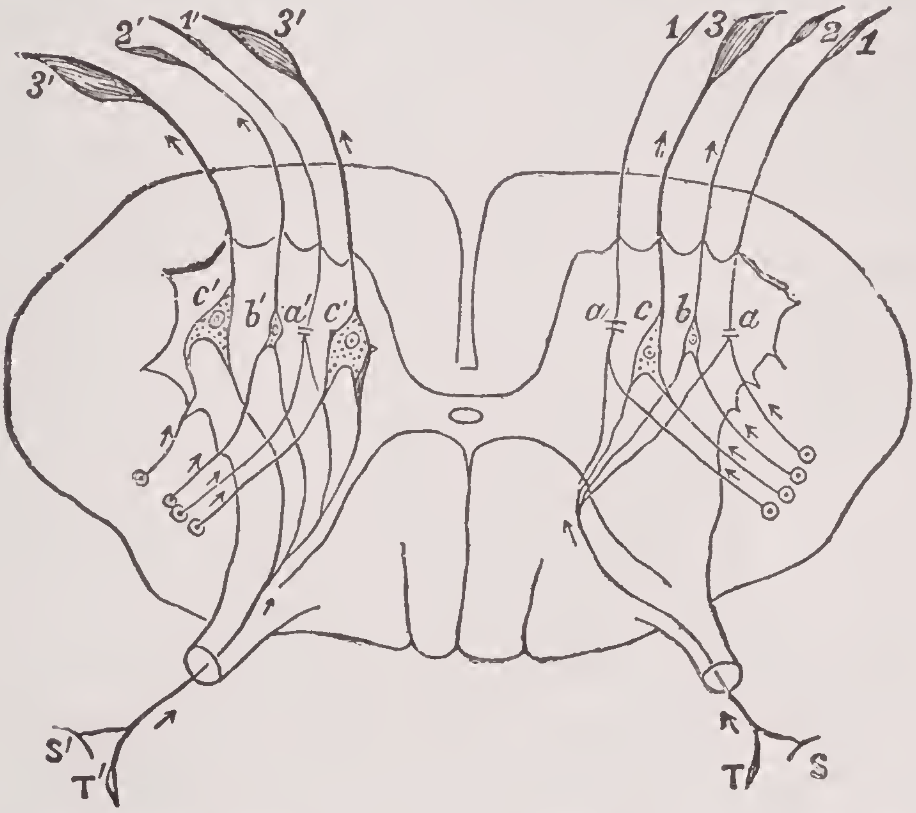

FIG. 60.—Diagrammatic representation of the symptoms that result from slow destruction of the multipolar nerve-cells of the anterior cornu (Bramwell). On the left side the disease is in an early stage. One nerve-cell (*a'*) is completely destroyed. Its muscular fibre (1') is completely atrophied. Voluntary motor and reflex motor impulses are "blocked" at the seat of the lesion (*a'*). One nerve-cell (*b'*) and its muscular fibre (2') are very much atrophied, but feeble motor and reflex impulses can still pass through the cell to the muscle. Two nerve-cells (*c'c'*) are healthy. Their muscular fibres are of normal bulk, and can be made to contract either by voluntary or reflex impulses. On the right side the disease is much more advanced. The muscular area is three-fourths degenerated. There is a total "block" at *a* and *a*. This condition represents a late stage of progressive muscular atrophy. The atrophy of the muscular fibres is represented as *simple*.

muscle to suffer, and its atrophy may present a deformity of contour closely resembling a subglenoid dislocation of the humerus.

In 90 per cent. of the cases the disease begins in the hand or shoulder. More exceptionally the affection may first show itself in the lumbar or abdominal muscles, and still more rarely in the muscles of the legs. The atrophied muscles are flaccid, and usually retain their normal electric reactions, for what muscle is left is good. In late cases, however, with rapid atrophy the R. D. may finally appear. Reflexes are diminished according to the amount of the atrophy. Fibrillary twitchings are not infrequently present. Atrophy proceeds from muscle to muscle in the order of the juxtaposition of their nerve-nuclei in the cord. Bilateral symmetry is usually preserved, although the wasting may be more marked on one side than the other. Owing to the contracture of unparalyzed muscles various deformities result; the "griffin-claw," or "main de griffe," has already been described. When the lumbar muscles are implicated the back is arched and the line of gravity falls behind the sacrum. When the abdominal muscles are affected the back is also arched, but the line of gravity falls in front of the sacrum.

In course of time all the voluntary muscles may become implicated, so that the patient is practically reduced to "skin and bone." The normal appearance of the facial muscles is in strong contrast to the wasting of the rest of the body. Ophthalmoplegia externa and bulbar paralysis may develop toward the close of the disease.

The **diagnosis** of "main de griffe" from lesion of the ulnar nerve is made by the absence of sensory symptoms. The diagnosis from idiopathic muscular atrophy is to be made by the different order of distribution of the atrophies in the latter disease, and by the fact that the latter condition usually occurs in young children. Amyotrophic lateral sclerosis is to be excluded by the absence of spasm and rigidity.

Prognosis.—The course of the disease is usually progressive. Death may result from exhaustion, from bulbar paralysis, or from respiratory affections. The disease may become quiescent at any time, and the patient may live for years. These cases are often seen as freaks in museums, under the name of "living skeletons."

Treatment is of no avail. The general nutrition of the muscles should be maintained by massage and electricity, and strychnine, arsenic, and nitrate of silver may be given internally. If the muscles of respiration be affected, extraordinary care should be taken to avoid cold, as even a trifling bronchitis may terminate fatally.

AMYOTROPHIC LATERAL SCLEROSIS.

Etiology.—This condition is really one of progressive muscular atrophy in which sclerosis in the lateral columns is sufficiently marked to produce symptoms, and is not at present regarded as an independent disease. The etiology is therefore the same as that of progressive muscular atrophy, except that the disease may occur after the age of seventy as a senile change.

Pathology.—The lesions are the same as those of progressive muscular atrophy, except that the direct and crossed pyramidal tracts show degeneration and sclerotic change, and the motor nuclear cells of the pons and of the medulla are more frequently involved.

The **symptoms** result from the combination of progressive muscular atrophy and lateral sclerosis. Wasting and weakness progress, with rigidity, contractures, and exaggerated reflexes. In the hands and arms the atrophy and weakness are especially well marked, while in the legs typical spastic paraplegia develops early. Fibrillary twitchings of the muscles are commonly observed. There are no sensory disturbances, and the bladder and the rectum are under full control. Ultimately the rigidity and muscular wasting become general, paralysis results, and symptoms of bulbar paralysis or of ophthalmoplegia externa make their appearance.

The **prognosis** is not as good as in progressive muscular atrophy, as the disease is seldom if ever arrested. Death results in from one to four years.

Treatment is of no avail. Long-continued rest in bed has been recommended.

BULBAR PARALYSIS.

Synonym.—Glosso-labio-laryngeal paralysis.

Two forms of bulbar paralysis are recognized—the acute and the chronic.

1. *Acute bulbar paralysis* results (1) from small hemorrhages into the medulla; (2) from areas of softening following embolism or thrombosis; (3) from an acute inflammatory affection analogous to anterior poliomyelitis; (4) as a terminal lesion of Landry's paralysis. The lesion is almost regularly bilateral.

The **symptoms** are those of the chronic form, differing only in their acuteness of onset. There may be hemiplegia or crossed facial paralysis. These acute cases prove rapidly fatal, although in rare instances the condition may become chronic.

2. *Chronic bulbar paralysis* is commonly associated with similar degeneration of the motor cells of the spinal cord.

The condition is rare before the age of forty, and usually appears as the terminal event of progressive muscular atrophy, of amyotrophic lateral sclerosis, or of Duchenne's paralysis.

Symptoms.—There is a progressive palsy of the tongue, lips, palate, pharynx, and laryngeal muscles, resembling in character the muscular changes of progressive muscular atrophy. The tongue becomes tremulous and is protruded with difficulty, and finally the lingual and dental consonants *d*, *l*, *n*, *r*, and *t* cannot be pronounced. The tongue atrophies. Chewing and swallowing are impaired. The lips become paralyzed and tremulous, the patient cannot whistle, and the lip-letters *b*, *f*, *v*, *o*, and *u* cannot be pronounced. Saliva drools from the mouth. There may be an emotionless expression, from facial paresis and atrophy. The pharynx becoming paralyzed, food regurgitates or cannot be swallowed. The laryngeal muscles waste, so that the voice is weak, almost inaudible, but extreme degrees of abductor paralysis are rare. Cough becomes impossible, and, the larynx being unprotected, aspiration- or deglutition-pneumonia is rendered possible. There are no sensory symptoms, and the mind is clear though emotional. Taste is not impaired. Pulmonary and cardiac crises occur when the nucleus of the vagus is affected. Severe dyspnœa appears on exertion, and, later, furious spontaneous attacks of suffocation, with cyanosis and a sense of extreme fulness in the chest, may appear.

The cardiac crises consist of a rapid and excessively feeble heart-action, pallor, anxiety, and a sense of impending death.

The **diagnosis** is not usually one of difficulty. The condition, however, may be simulated by a bilateral lesion in the lower portion of the third frontal convolution ("pseudo-bulbar paralysis of cerebral origin").

Prognosis.—The duration of the disease is from one to four years, with periods of temporary inactivity. Death results from inanition, from aspiration-pneumonia, or from heart-failure during a cardiac crisis.

Treatment.—The only thing that can be done is to advise the feeding of the patient by the stomach-tube when deglutition becomes impaired.

OPHTHALMOPLEGIA.

This rare disease is due to the progressive atrophy of the nuclei of the cranial nerves of the eye or the eyeball. According to whether the external or the internal muscles are affected, there are described ophthalmoplegia externa and ophthalmoplegia interna.

Ophthalmoplegia externa may be found associated with general paresis, progressive muscular atrophy, and locomotor ataxia, or the nuclear degeneration may be due to the pressure of tumors or to basilar meningitis. Mental disorders are present in about one-fifth of the cases, and atrophy of the optic nerve may coexist. There is a gradual loss of power in the extrinsic muscles of the eyeball, starting first in the levator muscles of the lid and in the superior recti. The loss of power finally becomes absolute. Ptosis, squint, and double vision appear during the earlier stages, but later the eyeball becomes fixed and immobile and may protrude. The disease is essentially chronic.

Ophthalmoplegia interna causes loss of pupillary reflex and of power of accommodation, and is usually associated with locomotor ataxia or with general paresis. The condition may be combined with the external form. Ophthalmoplegia interna may result from nuclear degeneration or from disease of the ciliary ganglion.

LATERAL SCLEROSIS (SPASTIC PARAPLEGIA).

Lateral sclerosis may be primary or secondary.

Primary lateral sclerosis is so rare a condition that by some its existence is doubted. It is said to be induced by syphilis, over-work, exposure, and sexual excesses, and to attack males of middle life.

Secondary lateral sclerosis is the most frequent form of spastic paraplegia, and results from any lesion destroying the motor cells in the cerebral cortex or cutting off the pyramidal fibres from their trophic centres in the motor cortical cells. These lesions may be cerebral or spinal. Such lesions are—(1) Tumors, softening, or hemorrhage in the brain, destroying the motor tract in one hemisphere. Secondary sclerosis is found below the lesion in the anterior median column and in the opposite lateral pyramidal tract. (2) Congenital malformation, hydrocephalus, or bilateral meningeal hemorrhage affecting both motor tracts. In these cases the descending degeneration is bilateral, involving both anterior median columns and crossed pyramidal tracts. (3) Any lesion in the cord, unilateral or bilateral, that separates the fibres of the motor tract from their trophic centre in the motor cortex of the brain. Such lesions are transverse myelitis, hemorrhage into the cord, or slow compression of the cord by tumor, disease, fracture, or dislocation of the vertebra, and meningeal hemorrhage. (4) The lateral columns may be involved with other tracts in the cord—with the cells of the anterior horns causing amyotrophic lateral sclerosis, with the posterior columns causing ataxic paraplegia. These combined lesions will be individually described.

Pathology.—The lesion is found to be limited to the lateral motor tracts. The anterior median columns may be involved in the case of a primary lesion in the brain. There is an increase in the connective-tissue framework, and a destruction and disappearance of the axis-cylinders of the nerve-fibres.

Symptoms.—*Of the Primary Form.*—There are first noticed a loss of endurance in walking, and stiffness and rigidity of the muscles. The muscular weakness is progressive, and merges into paralysis of voluntary motion.

Coincident with the weakness there is a rigidity of the affected limbs from a more or less continual spasm of the muscles. From time to time clonic spasms occur, especially during the night, during which the legs are twitched or suddenly jerked out. The gait becomes "spastic"—the toes stick to the ground, the knees touch or overlap in walking, and the leg is in a condition of spasmodic tension, or even shows distinct clonus when the ball of the foot rests upon the ground. The trunk is usually thrown forward by tonic contractures of the calf-muscles, so that crutches or canes held far in advance of the body become necessary. The power of locomotion is finally lost. The affected muscles do not atrophy, and the R. D. is not present. The reflexes are regularly increased. The knee-jerk is excessive and prolonged, and may be radiated to the arms or to the opposite leg. Ankle-clonus is easily obtained. There are no essential sensory symptoms, though there may be dull pains in the muscles, and the bladder and rectum are not usually involved until late in the disease. Ocular symptoms are rare. The arms may escape altogether, or rigidity may appear as a late manifestation of the disease.

Of the Secondary Form.—The symptoms are bilateral or unilateral according as to whether one motor tract or both are affected. Cerebral lesions usually lead to unilateral sclerosis. The symptoms appear rapidly or gradually according to the nature of the primary lesion. "Late rigidity" with hemiplegia following cerebral hemorrhage is synonymous with secondary lateral sclerosis.

Spastic paraplegia of infants is usually a birth-palsy due to meningeal hemorrhage. In other cases the condition arises from an arrested development of the pyramidal tracts. The symptoms may be bilateral, and the arms are not infrequently involved. Idiocy, imbecility, and other mental defects are usually present.

The **diagnosis** from hysterical spastic paraplegia may be one of great difficulty, as the hysterical form may exactly reduplicate the symptoms of the organic disease. In favor of the hysterical condition are (1) the sudden development of symptoms, (2) the history of hysterical attacks in the past, (3) the presence of anæsthesia, hyperæsthesia, or other hysterical manifestations, and (4) the sudden remission of muscular contractions, which in the organic form should be more permanent.

Prognosis.—The course of the disease is chronic, extending over many years. The disease is the least fatal of all the chronic spinal affections. Its progress may be arrested at any time.

Treatment.—In syphilitic cases mercury and iodide of potassium may be tried; strychnine is contraindicated; the bromides may be of use in reducing the condition of over-reflexes; otherwise there is no medicinal treatment of any value.

LOCOMOTOR ATAXIA.

Etiology and Synonyms.—This disease occurs in males ten times as frequently as in females, and is most common between thirty and fifty years of age. Its occurrence under the age of twenty-five is rare. The great predisposing cause is syphilis, which precedes the disease in two-thirds of the cases. Among exciting causes are sexual excesses, great physical exertion, and repeated exposures to wet and cold. *Synonyms:* Posterior spinal sclerosis; Tabes dorsalis.

Pathology.—There is a sclerosis, beginning first in the middle zone of the column of Burdach and in the column of Lissauer (the narrow marginal zone lying between the posterior horn and the pyramidal tract), and extending to the column of Goll and the remainder of the column of Burdach (Fig. 61). The network of fibres about the vesicular columns of Clarke are affected by the sclerotic processes early in the disease. The lesion begins in the lumbosacral region and extends upward throughout the cord. In long-standing cases the sclerosis extends to the lateral columns and leads to the degeneration of the pyramidal and direct cerebellar tracts. The posterior nerve-roots show the lesions of a degenerative neuritis, and become small and atrophic. These nerve-changes are frequently first observed in the cutaneous filaments, and may even precede the sclerosis of the posterior columns. The meninges over the lateral and posterior columns are thickened and abnormally adherent; their blood-vessels show the changes of arterio-sclerosis. Besides neuritis there may be muscular atrophy. Atrophy may occur in any of the cranial nerves, especially the optic, third, auditory, and pneumogastric nerves. There may be sclerosis of the restiform bodies or of the inferior peduncles of the cerebellum, or sclerotic areas in the hemispheres may resemble the lesions of general paresis.

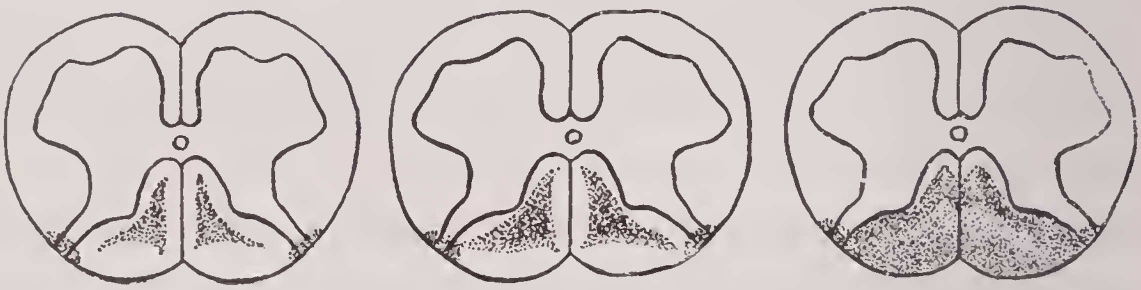

FIG. 61.—Localization of the lesion in successive stages of locomotor ataxia.

The disease is not yet thoroughly understood, and three theories are advanced to account for the lesions: (1) That there is a primary sclerosis of the posterior columns; (2) that the sclerosis is dependent upon arterial degeneration of the vessels entering the posterior root-zones; and (3) that the disease originates in the ganglia of the posterior nerve-roots, with secondary degeneration of the sensory fibres entering the cord from these ganglia, with secondary sclerotic changes in their course.

The **symptoms** of locomotor ataxia may be divided into three stages: (1) The stage of pain; (2) that of ataxia, and (3) that of paralysis.

1. *Stage of Pain.*—The following are the characteristic symptoms of this pre-ataxic stage:

(*a*) *Pain*, of a paroxysmal darting character, appears in 90 per cent. of the cases. The pains are bilateral, dart down the legs, and are generally referred to the deeper structures of the limb. Their distribution is irregular, rarely corresponding to the nerve-trunks. The characteristics of the pains are their "lightning" character and the absence of local tenderness. Occasionally trophic eruptions appear. In rarer instances the pain may be diffused and superficial, or the feeling may be described as one of intense heat or cold. Lightning pains in the arms occur when the lesion extends to the cervical enlargement of the cord. Pain may be absent or insignificant in a few cases beginning with rapid optic atrophy, and in these patients ataxia is not apt to be developed.

(*b*) There are symptoms of perverted sensation. The patient may complain of numbness or tingling in the legs or the feet, or may feel as though he were walking on cotton or on air-bags instead of on solid ground. There may be a sensation of tightness and pressure about the waist. In some instances the "muscular sense" becomes so impaired that the patient cannot tell in what position the limbs are placed when the eyes are shut. During this first stage, however, the sensory symptoms are subjective, and areas of anæsthesia or of retarded sensation cannot be demonstrated.

(*c*) *Loss of knee-jerk* (Westphal's symptom) is one of the earliest indications of the disease, and its association with the lightning pains and the ocular symptoms forms a symptom-group absolutely diagnostic of locomotor ataxia. The superficial reflexes remain good.

(*d*) *Ocular Symptoms.*—There may be ptosis, strabismus, double vision, or in rare cases ophthalmoplegia externa. Contracted pupils (" myosis spinalis ") are frequent, but not constant. Optic atrophy may develop, causing dimness and restricted field of vision and color-blindness, and the atrophy may progress until the vision is entirely lost. Cases in which the atrophy of the optic nerve appears early and progresses rapidly do not seem to develop the second stage of ataxia. The Argyll-Robertson pupil occurs during the first stage in over 80 per cent. of the cases. In this condition the pupils do not react to light, but accommodation to distance is preserved.

(*e*) *Bladder and Rectum.*—There may be lightning pains referred to these viscera. Constipation is usually obstinate. Micturition may be frequent and painful, or there may be imperfect control of the bladder, with dribbling, partial retention of urine, and cystitis. Impotence may appear, occasionally preceded by priapism and sexual excitement.

2. *Stage of Ataxia.*—Old symptoms persist while new symptoms appear. The lightning pains may continue, but they tend to become less and less severe. Objective sensory disturbances can now be demonstrated; there may be areas of anæsthesia or hyperæsthesia or of retarded sensation. The power of localizing pain may be lost, and the muscular sense becomes more and more impaired, so that motions cannot be made accurately without the aid of sight; hence a blind ataxic patient may become almost totally helpless. The eye-symptoms noted above continue, or they may appear for the first time. Optic atrophy occurs in 20 per cent. of the cases, and its antagonism to the development of ataxia has already been noted. There is usually difficulty in emptying the bladder, and retention with cystitis is apt to

result. Deafness may occur from neuritis of the auditory nerve. The characteristic symptoms of the second stage are ataxia, visceral crises, and trophic changes.

Ataxia usually develops in the legs, although in rare instances the arms may be first involved. The patient loses the power to co-ordinate muscular movements so as to produce a harmonious result There is an inability to stand steadily with the eyes shut (Romberg's symptom), to walk readily in the dark, or to turn quickly without falling. Inco-ordination of the arms is usually apparent in writing, in buttoning the clothes, or in handling the knife and fork when at table. Ataxia is demonstrated by having the patient stand or walk with the eyes shut, or touch toe to heel or heel to knee, or to rapidly touch the nose with the finger when the eyes are shut. The gait becomes characteristic. The legs are far apart; the body is inclined forward, so that the support of a cane may be a necessity. The foot is lifted high at each step, and is planted forcibly upon the ground with a stamp or a slap. The muscular power is maintained, and the nutrition of the muscles, except toward the close, is usually unimpaired.

Visceral crises are characterized by paroxsymal pain in the various viscera. Thus, gastric, laryngeal, renal, cardiac, rectal, and genital crises are described, of which the gastric and the laryngeal are the most common, and are due to neuritis of the pneumogastric nerve. A gastric crisis consists of severe paroxysmal pain, vomiting, and hyperacidity. There may be hæmatemesis. The laryngeal crisis gives rise to dyspnœa, hoarse coughing, and intense pains in the shoulder and spine. There may be fatal asphyxia, or the larynx may become anæsthetic, so that death may result from aspiration-pneumonia. Renal crises may reproduce the symptoms of calculus. Cardiac crises give rise to pain in the heart, irregular and feeble pulse, and syncope.

Trophic Changes.—Of these changes, the most common are the arthropathies or joint-lesions known as "Charcot's joints" (Fig. 62), which occur in from 5 to 10 per cent. of

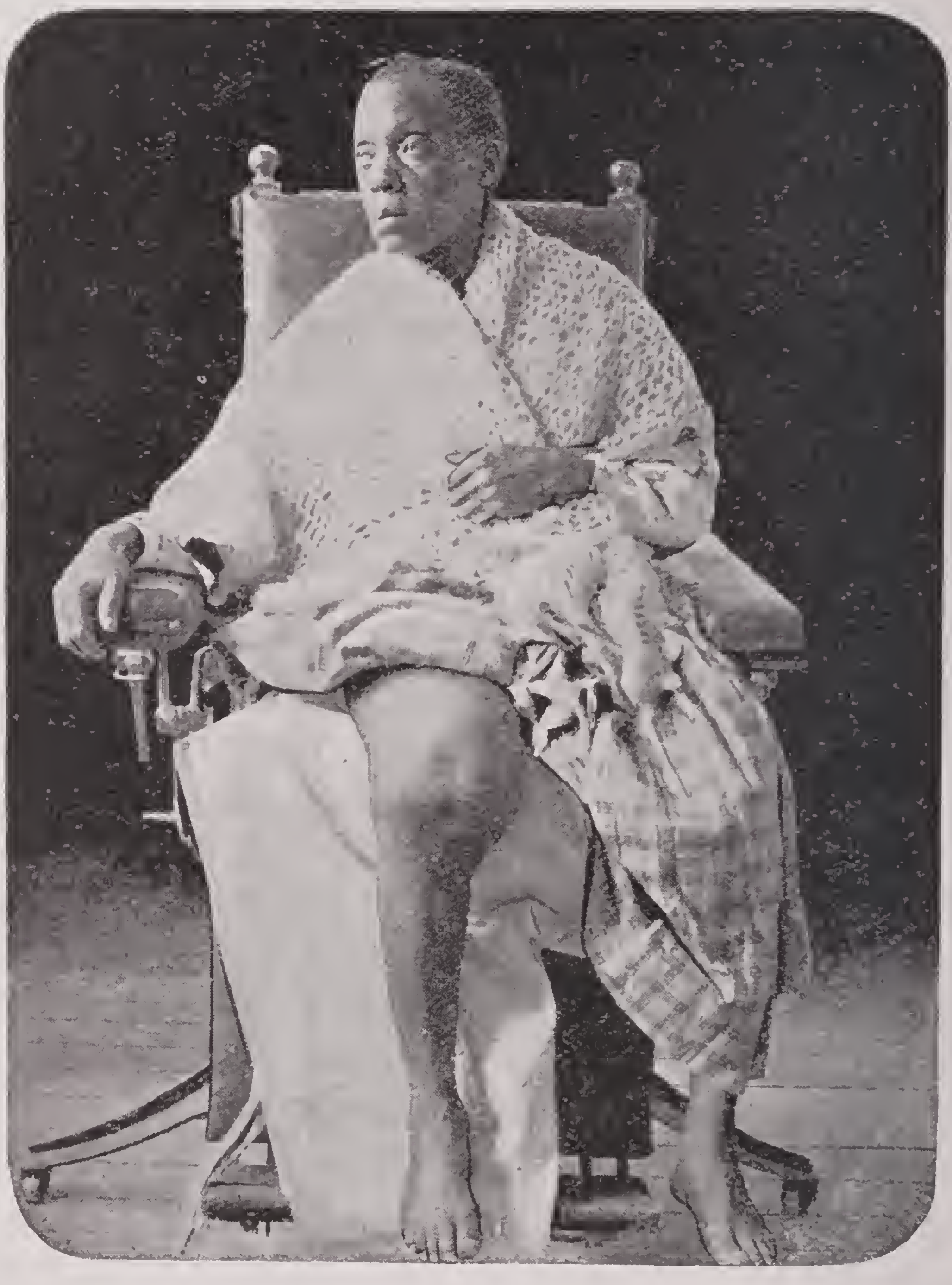

FIG. 62.—Locomotor ataxia, showing Charcot's knee (personal observation).

all cases. These changes are most common in the larger joints, especially the knee. The joint swells rapidly from serous effusion, the articular ends of the bones become absorbed, ligaments soften so that dislocations and unnatural mobility become evident, and there is an irregular production of new bone about the edges of the articular surfaces. These changes are essentially trophic in character, but an exciting cause may be found in some traumatism of which the patient is unconscious owing to the anæsthesia of the parts. The joint-lesions progress without fever and without pain, and the symptoms may develop in from twenty-four to forty-eight hours.

The chief points of **differential diagnosis** from rheumatoid arthritis are as follows:

Rheumatoid Arthritis.	*Charcot's Joint.*
Hypertrophy.	Atrophy.
Painful.	Painless.
Limited mobility.	Increased mobility.
Slow process.	Rapid process.
Small joints.	Large joints.
Symmetry of lesions.	No symmetry; usually unilateral.
No ataxia.	Ataxia.

Besides Charcot's joint other trophic changes may occur. The bones may rarefy and be the seat of spontaneous fracture. Absorption of the articular ends of the bones leads to dislocation. There may be herpes, œdema, local sweating, perforating ulcer of the foot, inflammation and falling of the nails, and atrophy of muscles.

3. *The stage of paralysis* occurs when the patient loses the power of walking. Paraplegia develops from involvement of the lateral columns. The patient may develop during the second or third stage of the disease general paresis, melancholia, or delusional insanity. Cystitis and pyelo-nephritis are apt to develop. Pneumonia or bedsores may hasten the final issue.

Prognosis.—The course of the disease is chronic, lasting from twenty to forty years. Ataxia is rarely developed until from five to eight years after the beginning of the disease. There have been described rare instances of acute ataxia in which the patient became bedridden within a few months. Recovery never occurs, although the disease may be arrested at any time, especially during the first stage, and may even show periods of temporary improvement. The disease itself seldom causes death.

Treatment.—A quiet and regular mode of life should be enjoined. Alcoholic and sexual excesses should be absolutely interdicted. Rest in bed for several months is sometimes serviceable in modifying the neuralgic pains. Spinal douches, tepid or cool, may be ordered daily, but extreme temperatures should be avoided.

The medicinal treatment is somewhat varied, as there seems to be no drug capable of exerting a beneficial effect upon the disease to any appreciable extent. The drugs that have been recommended are mercury and iodide of potassium, especially in recent syphilitic cases; arsenic in full doses; nitrate of silver in gr. $\frac{1}{4}$ doses three times daily for periods of not longer than two months; chloride of sodium and gold; chloride of aluminum in 2- to 4-grain doses; and ergot in moderate doses.

The pains may be relieved by counter-irritation to the spine, preferably by the thermo-cautery applied every two or four weeks, but the application of counter-irritants should not be severe, especially over anæsthetic portions of the skin, as destructive trophic changes may ensue. Pain may also be relieved by phenacetine or antipyrine, but opium should be given with caution, for fear of the habit being formed.

The treatment by suspension is now being abandoned, as the published results do not agree with the first enthusiastic reports. Charcot's joints are to be treated by rest and apparatus. Morphine may be indicated during the visceral crises.

HEREDITARY ATAXIA.

Etiology and Synonym.—The disease may or may not be hereditary; in the latter case a history of nervous disorders—insanity, inebriety, or nervous irritability—is generally obtained. The disease, which is apt to appear between the fifth and fifteenth years, rarely as late as the twentieth year, is one of defective development. *Synonym:* Friedreich's ataxia.

Pathology.—There is extensive sclerosis of the posterior and lateral columns of the cord; this sclerosis may extend upwards to involve the medulla.

Symptoms.—Ataxia is first developed in the legs, but the gait differs from that of locomotor ataxia in being more swaying and irregular and less stamping. Romberg's symptom may or may not be present, and the reflexes may be preserved. Ataxia appears in the arms, giving rise to irregular choreiform movements. Rhythmical movements may also be observed during rest. Nystagmus and slow, scanning speech are commonly observed, but visceral symptoms and optic atrophy are uncommon. Trophic changes are not observed. There is a fairly characteristic deformity of the foot; the patient walks on the outer edge of the foot, the big toe is flexed dorsally upon the first phalanx, and talipes equinus is developed. There are no sensory symptoms. The mind becomes impaired late in the disease. As the disease progresses paralysis appears; this paralysis may become complete.

Prognosis.—The disease is incurable, but its course extends over years.

Treatment is unavailing.

ATAXIC PARAPLEGIA.

Etiology.—Males of middle age are most frequently affected. There may be a history of exposure to cold or of sexual excesses, but antecedent syphilis is rarely to be demonstrated.

Pathology.—The lesion consists of a combined sclerosis of the posterior and lateral columns, beginning in the lumbar region. The nerve-roots are not involved as in locomotor ataxia.

Symptoms.—There are slowly developing weakness and rigidity of the legs, with ataxia. The knee-jerk is exaggerated, and ankle-clonus can easily be obtained. The Romberg symptom is generally well marked. A dull, aching pain in the sacral region is the only sensory symptom of importance. Eye-symptoms are rare. The ataxia and weakness may extend to the arms, and in many cases there may be developed mental symptoms resembling those of general paresis. The muscular weakness ultimately merges into paralysis.

Prognosis.—The disease is incurable. Death results from complications rather than from the disease itself.

The **treatment** is that of chronic myelitis.

MYELITIS, ACUTE AND CHRONIC.

Etiology.—Myelitis may occur (1) from excessive physical exertion, from exposure to wet and cold, or from sexual excesses; (2) from injury or disease of the vertebral bones causing compression or destruction of the spinal cord, or from tumors of the cord itself; (3) from acute infectious diseases, especially small-pox, measles, and typhus fever; (4) syphilis as an exciting or predisposing cause of myelitis is questionable.

Pathology.—The affected area of the cord feels soft and may even be diffluent. The softened area may be grayish or reddish in color (" red" or " gray softening ") according to whether or not small hemorrhages into the cord-substance have occurred. The nerve cells and fibres swell, undergo fatty degeneration, and the myelin oozes out as fatty droplets. Large numbers of inflammatory corpuscles are everywhere present, and "Deiter's spider-cells," due to proliferation of the neuroglia, are to be seen. The laminated bodies known as "corpora amylacea" are also present. The blood-vessels are dilated and may rupture. The meninges may also be involved. After a time conservative changes assert themselves. The area becomes firm from an increase of connective tissue, so that the cord at the affected point becomes converted to a mass of cicatricial tissue containing perhaps a few nerve-fibres and cells. To this condition the name of "chronic myelitis" is applied.

Secondary degenerations result (1) in the lateral columns below the lesion, and (2) in the posterior column and direct cerebellar tract above the lesion.

The affected area varies. In *general* myelitis the cord is involved along its entire length; in *disseminated* myelitis various segments of the cord at different levels are affected; in *transverse* myelitis one or two segments of the cord at one level are destroyed.

The effects of a transverse lesion of the cord are—(1) Voluntary motion is cut off from the parts below the lesion—paralysis. (2) Sensation is cut off from the parts below—anæsthesia. (3) Inhibitory fibres from the motor cortex checking over-reflexes are destroyed at the site of the lesion—increased reflexes. (4) The nutrition of the parts supplied directly from the affected area is impaired, atrophic changes resulting in nerve, muscle, and skin.

Symptoms.—Four stages are described: (1) A stage of premonition, (2) one of irritation, (3) one of destruction, and (4) one of descending degeneration.

1. *Premonitory Stage.*—There may be peculiar sensations in the parts afterward to be more seriously affected, and motor weakness may be noticed. There may be the

"girdle sensation" of a string tied about the waist, from irritation of the nerves at the upper level of the lesion. In other cases these symptoms are absent, or there may be only a chill and fever.

2. The *irritative stage* is of short duration. Sensory symptoms consist of hyperæsthesia and neuralgic pains below the lesion, and the girdle-sensation. Pain in the back is uncommon unless the meninges become inflamed. The sensory symptoms of irritation soon become admixed with those of destruction, and feelings of numbness and areas of anæsthesia appear. The motor symptoms consist of twitchings, cramps, and spasms, combined with some loss of voluntary power. The duration of this stage varies from several hours to one or two days.

3. *Stage of Destruction.*—Two groups of symptoms are recognized—one, *direct*, due to the destruction of cord-tissue, and one, *indirect*, due to the cutting off of impulses to and from the brain.

Direct symptoms are observed in the parts supplied directly from the affected segments. There is muscular paralysis, with atrophy and the reaction of degeneration. Reflexes are lost. There is anæsthesia of the skin supplied by the affected spinal nerves, with vaso-motor symptoms (coldness, sweating) and atrophic changes, as bed-sores. The atrophic bed-sores are deep and gangrenous. The distribution of the direct symptoms depends upon the extent of the lesion. In general myelitis they are universal: if disseminated, they are scattered; if transverse, they are limited to one level, the arms being involved in cervical myelitis, the trunk in dorsal myelitis, the legs if the myelitis involve the lumbar enlargement.

Indirect symptoms result from the severance of the motor pyramidal and the sensory fibres at the site of the lesion.

Below the lesion there is paralysis, with increased reflexes and muscular rigidity. The paralyzed muscles do not atrophy, and there is no reaction of degeneration. If the myelitis involve the lumbar enlargement, *direct* symptoms may be present in the legs—paralysis, atrophy, loss of reflexes, and the reaction of degeneration.

If the reflex centre for the bladder be destroyed, the bladder will no longer contract to expel its contents, but there will be incontinence from over-distention. Usually, however, the lesion is higher up than this, so that the reflex bladder-centre is still intact; there will then be reflex and unconscious passage of urine. The danger of cystitis threatens every case of myelitis. The functions of the rectum are similarly affected.

There is regularly anæsthesia below the lesion. The imperfect sensation allows of the formation of bed-sores, from pressure or from dirt, over the sacrum, the glutei, or the heels. These pressure bed-sores are at first superficial, and can be prevented by careful nursing. The atrophic bed-sores in areas of skin supplied by nerves from the destroyed segment cannot be prevented, and they are large, deep, and gangrenous.

Cerebral symptoms are rare. There may be optic neuritis with blindness. The pulse varies from 100 to 140; the temperature varies between 102° and 104° F. The fever quickly subsides unless cystitis, pyelitis, or acute atrophic bed-sores develop. During this stage the patient may die from paralysis of the respiratory muscles, pneumonia, cystitis, pyelitis, suppurative nephritis, or acute atrophic bed-sores. The majority of patients, however, pass into the stage of descending degeneration, or "chronic myelitis."

4. *Stage of Descending Degeneration.*—In a few cases some motor power is regained and some sensations are perceived, so that the patient is able to get about on crutches, although with spastic paraplegia and loss of bladder-

control. In other cases no improvement is noted, and the patient remains bedridden, with paralyzed, twitching limbs and cystitis. Pain in the back develops in the majority of cases, from the occurrence of chronic meningitis. Death results from suppurative nephritis, pneumonia, or bed-sores.

Prognosis.—In very acute cases death may result in five or ten days. Transverse myelitis in the cervical region is usually fatal from paralysis of the respiratory muscles. The majority of cases, however, pass into the chronic condition of spastic paraplegia, from which but trifling improvement can be expected.

Treatment.—During the earlier stages the patient should lie upon the side or face while the spine is cupped or is covered with a Chapman ice-bag. Active purgation by calomel or salts is indicated at the onset. Ergot in large doses has been recommended, but not too much is to be expected from its use. Morphine may be necessary for the relief of the pain. Great care should be exercised to prevent bed-sores and cystitis. The sheets should be drawn smooth, without wrinkles, and should be kept free from crumbs. The skin of the back is best hardened by daily frictions with alcohol and alum-water. The bed should also be kept dry, pads of absorbent cotton or a urinal being placed in position for the incontinence of the urine. Carefully padding the patient with small pillows may prevent a bed-sore, should a pressure-point become red and chafed, while in many cases a water-bed is indispensable. When bed-sores occur, simple antiseptic dressings are indicated. For the prevention of cystitis the urine should be drawn at regular intervals. The catheters should be kept surgically clean. If cystitis develop, the bladder should be washed out daily with a boric-acid solution (ʒj : Oj).

For the chronic condition counter-irritation of the spine has been advised, but blistering agents should never be used, as there is danger of bed-sores developing. Spasm of the limbs is relieved by heat to the spine or by general hot baths. Massage is indicated to keep up the nutrition of the muscles. Drugs are of no value in myelitis. Potassium iodide and mercury may be given to syphilitic subjects, and phosphorus and arsenic may be employed as nerve-tonics. Nitrate of silver (gr. $\frac{1}{4}$) is often recommended. Strychnine is contraindicated if spasm exist in the paralyzed muscles.

ACUTE ASCENDING PARALYSIS.

Etiology and Synonym.—The disease is most common in men between the ages of twenty and thirty. Some cases have followed infectious fevers. *Synonym:* Landry's paralysis.

Pathology.—In many of the cases an interstitial neuritis of the nerve-roots has been demonstrated, so that the disease has been classed as a peripheral neuritis. In other cases, however, no lesions have been found, so that it is supposed that the paralysis is due to some form of microbic poisoning. The disease bears a close resemblance to paralytic rabies.

Symptoms.—Weakness in the legs merges within a few hours into paralysis, which spreads to involve the trunk, arms, and neck. Finally the muscles of respiration, deglutition, and articulation become affected, and there may be facial and eye-palsies. The reflexes are lost, but the muscles neither waste nor show the electrical reactions of degeneration. There may be numbness, tingling, or hyperæsthesia, but sensory symptoms are neither constant nor essential. The bladder and rectum are seldom involved. Febrile reaction is trifling.

Prognosis.—Death may result within two days or may be postponed for one or two weeks. Recovery has occurred only in rare instances.

Treatment.—Ergotin in 2-grain doses may be given every hour, and success has followed its administration; sodium salicylate and benzoate also have been recommended; otherwise the treatment is symptomatic.

SYRINGO-MYELIA.

Etiology.—This rare disease is probably of congenital origin. Its exact cause is unknown. The symptoms usually appear in males between the ages of fifteen and twenty-five.

Pathology.—There is a development of embryonal neuroglia-tissue about the central canal of the spinal cord extending to involve the entire central gray matter. Degeneration and liquefaction result in the formation in the spinal cord of a cavity filled with cerebro-spinal fluid; the walls of the cavity are composed of gliomatous tissues. The usual situation is in the lower cervical and upper dorsal region. In other cases the cavity extends the entire length of the spinal cord. The cavity may invade the anterior horns, causing the symptoms of chronic anterior poliomyelitis, or may invade the posterior horns and columns, causing the symptoms of posterior sclerosis.

The **symptoms** begin insidiously about the time of adolescence, and extend over years. There are aching pains in the neck and the arms, followed by muscular atrophy, first in the hands, then in the arms and trunk. There is a loss of the sensations of temperature and pain, but the touch-sensations remain intact—a form of partial anæsthesia which is almost pathognomonic. The legs become involved late in the disease, and show the symptoms of spastic paraplegia. From the involvement of the spinal muscles, curvature (scoliosis) almost regularly results. Vaso-motor, secretory, and trophic symptoms are common in the affected parts—cyanosis, œdema, sweating, ulcers, bullæ, defective growth of the nails, brittleness of the bones. Felons are common. There is loss of control over the bladder and rectum if the lumbar region of the cord be involved.

The **prognosis** is bad, although the disease extends over years. The latter stages of the disease resemble chronic muscular atrophy. Death may result from involvement of the medulla.

Treatment is inoperative. Arsenic and nitrate of silver are generally given as a routine, but beneficial results are not to be expected from medication.

COMPRESSION-MYELITIS (SLOW COMPRESSION OF THE CORD).

Etiology.—The spinal cord may be compressed (*a*) by disease of the vertebral bones, especially caries; (*b*) by thickened membranes; (*c*) by tumors of the cord, membranes, or bones. The most common tumors are carcinoma, (usually secondary to primary growths in the breast), retroperitoneal sarcoma and aneurysm (causing erosion of the bodies of the vertebræ, so that they come to lie directly on the spinal cord itself), and sarcoma of the membranes.

The **pathology** is that of a pressure-atrophy.

The **symptoms** are those of chronic myelitis slowly developing with a prolonged stage of irritation.

Compression of the spinal-nerve-roots causes neuralgic pains with areas of anæsthesia (" anæsthesia dolorosa ") and with muscular spasms followed by paralysis, loss of reflexes, and atrophy of muscle.

Compression on a spinal segment gives rise to anæsthesia, paralysis, atrophy, loss of reflexes, and the reaction of degeneration in the muscles supplied directly from the compressed segment. Below the affected segment there are the symptoms of spastic paraplegia—paralysis, increased reflexes, absence of atrophy and of the reaction of degeneration. There is loss of bladder- and rectum-control. If the compressed segment be in the lower dorsal and lumbar regions, the reflexes in the legs will be lost and the muscles will atrophy.

The **prognosis** depends upon the cause of the compression.

Treatment is that of the original cause. Caries of the vertebræ is best treated by suspension. Tumors may be removed by operation if it be practicable. In some cases of bone disease laminectomy may be performed with benefit.

TUMORS OF THE SPINAL CORD.

Etiology and Pathology.—Tumors of the spinal cord are rare and are usually secondary to growths elsewhere. Sarcoma and tubercular, syphilitic, and gliomatous growths are most frequently observed. The compression of the spinal cord leads to a chronic myelitis at the seat of the growth, and in rare cases may induce the condition of syringomyelia.

Symptoms.—The symptoms are those of a compression-myelitis or of Brown-Séquard's paralysis, of slow development and characterized by such extreme pain in the sensory areas corresponding to the segment in which the tumor grows that the name "paraplegia dolorosa" has frequently been applied.

The **prognosis** is bad in inoperable growths.

The **treatment** is to remove the tumor if possible; otherwise the treatment is symptomatic.

BROWN-SEQUARD'S PARALYSIS.

Etiology.—One half of the spinal cord may be destroyed by tumors, by hemorrhages, by disease of the vertebral bones, or by traumatism.

The **pathology** is that of a destructive lesion involving a lateral half of a spinal segment.

The **symptoms** are best appreciated by consulting the following table of Gowers:

CORD.

Zone of cutaneous hyperæsthesia. Zone of cutaneous anæsthesia.	Lesion.	
Motor palsy.		Muscular power normal.
Hyperæsthesia of skin.		Loss of sensibility of skin.
Muscular sense impaired.		Muscular sense normal.
Reflex action at first lessened, then increased.		Reflex action normal.
Temperature raised.		Temperature same as that above the lesion.

Treatment is directed to the cause of the hemi-lesion; otherwise the treatment is that of myelitis.

5. DISEASES OF THE CRANIAL NERVES.

OLFACTORY NERVE.

Anosmia, or loss of the sense of smell, may occur with chronic nasal catarrh or with diseases of the olfactory nerves or bulbs following meningitis, frontal tumors, or caries of the bones. The symptom is not uncommon among insane and hysterical patients.

Hyperosmia, or increased sensitiveness, and parosmia, or subjective perversions, of the sense of smell, are not infrequently observed in neurotic patients. Parosmia may precede an attack of epilepsy.

OPTIC NERVE.

Many of the diseases of the optic nerve clearly belong to the domain of ophthalmology, and therefore will not be considered here.

Destructive lesions in various parts of the optic tract produce the following results (see Fig. 55):

1. Lesions of the optic nerve produce blindness of the corresponding eye.
2. Lesions of the chiasm may produce blindness of both eyes (if the chiasm be totally destroyed), temporal hemianopia (if the central part of the chiasm be involved), or nasal hemianopia (if both lateral regions of the chiasm be affected).
3. Lesions of the optic tract produce lateral hemianopia.
4. Lesions of the cuneus produce lateral hemianopia.
5. Lesions of the angular gyrus give rise to hemianopia and mind-blindness, rarely to crossed amblyopia.

Hemianopia, though usually of organic origin, may occur with hysteria, migraine, and lithæmia. In hemianopia, if the pupil reacts when a ray of light is thrown upon the sensitive half of the retina, the lesion is in the optic radiation or in the cerebral cortex.

THIRD NERVE.

Nuclear lesions of the third nerve are usually associated with disease of the other ocular-nerve-centres (see Ophthalmoplegia). Disease of the third-nerve-trunk is not uncommon. The nerve may be the seat of a neuritis (especially with locomotor ataxia and after diphtheria), it may be compressed by meningitis, tumors, or aneurysms at the base of the brain, or it may be paralyzed from exposure to cold, from rheumatism, from syphilis, or by an attack of migraine. Paralysis of the third nerve gives rise to external strabismus, ptosis, dilatation of the pupil, loss of pupil-reflex and of accommodation to distance, and to diplopia, or double vision.

A form of oculo-motor palsy is described as occurring chiefly in women, and as recurring at intervals of several months, associated with pain and migraine.

Spasm of the muscles supplied by the third nerve is not uncommon in meningitis and in hysteria. Slow rhythmical oscillations of both eyeballs (nystagmus) occur in congenital and acquired brain affections and in albinism, and is not uncommon among coal-miners.

FOURTH NERVE.

The causes of fourth-nerve-paralysis are similar to those causing third-nerve-palsy. The symptoms are a slight convergent strabismus when the eye is rolled downward, and double vision when the patient looks down.

FIFTH NERVE.

PARALYSIS.—The nucleus may be involved by hemorrhages or tumors of the pons, or the branches of the nerve may be affected within the cranium by meningitis, caries, or tumors. The lower divisions are not infrequently involved by tumors of the upper jaw. Primary neuritis is rare.

Symptoms.—*Sensory.*—There is anæsthesia of the skin of the face and head, the conjunctiva, and the mucosa of the lips, tongue, soft and hard palate, and nose. The anæsthesia may be preceded by hyperæsthesia or by tingling feelings.

Motor.—The temporal and masseter muscles are paralyzed, and the jaw, when depressed, moves toward the paralyzed side. The motor palsy is usually due to lesions involving the trunk of the nerves.

Trophic and Vaso-motor Changes.—There may be ulcerations of the mucosa, falling of the teeth, opacity and ulceration of the cornea, diminished salivary, nasal, buccal, and lachrymal secretions, flushings and pallor, and herpes. The trophic changes occur if the Gasserian ganglion is affected.

Gustatory.—There is regularly loss of the sense of taste in the anterior two-thirds of the tongue.

Spasm of the Muscles of Mastication (Trismus; Lockjaw).—Trismus occurs with tetanus, tetany, and hysteria, from reflex irritation from diseases of the teeth and jaws, and from irritation of the motor nucleus of the fifth nerve. Trismus nascentium is caused by infection through the umbilicus of newly-born children. The spasm may be tonic or clonic. Clonic spasms may occur as a symptom of chorea.

NEURALGIA (Tic Douloureux).—Neuralgia of all the branches of the fifth nerve is rare; usually the ophthalmic, alone or with the superior maxillary division, is affected.

For the etiology of neuralgia see the article on Neuralgia.

Supraorbital neuralgia is marked by shooting pains along the course of the nerves, by painful spots at the supraorbital notch, at the inner angle of the orbit, and at the junction of the bone and cartilage of the nose. There are usually intolerance to light, injection of the conjunctiva, and increased lachrymation. The skin may be exquisitely tender to the touch. In severe cases there may be painful spasm of the facial muscles (spasmodic tic). In protracted cases the hair on the affected side may become gray.

Superior Maxillary Neuralgia.—Pain is referred to the teeth of the upper jaw, and a painful point is located at the infraorbital foramen.

Inferior Maxillary Neuralgia.—The pain is experienced about the ear and in the teeth of the lower jaw, and painful spots may be elicited along the auriculo-temporal nerve.

The treatment of neuralgia will be considered under a separate heading (see page 859).

SIXTH NERVE.

Abducens palsy occurs most commonly with syphilis and locomotor ataxia, and causes convergent strabismus, double vision, and inability to rotate the eye outward. When the nucleus of the nerve is involved, there is, in addition to the paralysis of the external rectus, an inability of the internal rectus of the unaffected eye to turn that eye inward. Both eyes are therefore deviated to the opposite side, away from the lesion (conjugate deviation).

SEVENTH NERVE—FACIAL.

Facial Paralysis (Bell's Palsy).—The innervating fibres of the facial muscles may be paralyzed (1) above the nucleus, (2) at the nucleus, or (3) after leaving the nucleus.

1. *Supranuclear paralysis* is caused by destructive lesions of the cortex, corona radiata, and internal capsule, and is, as a rule, accompanied by hemiplegia on the same side as the facial paralysis. Facial paralysis alone from cortical or central lesions is uncommon. Supranuclear paralysis differs from the peripheral form in that the electrical reactions of the affected muscles remain normal, and the upper branches of the facial nerve are not involved, so that the patient can wink and can corrugate the forehead.

2. *Nuclear paralysis* is uncommon. The nucleus may be involved by tumors, by hemorrhages, or by softening, or with diphtheria or anterior poliomyelitis. The symptoms are the same as those of the peripheral type. Lesions of the pons may cause facial paralysis on the same side, and crossed hemiplegia, the so-called "crossed facial paralysis" (See Fig. 57).

3. *Peripheral or Infranuclear Paralysis.*—The nerve-trunk may be involved (*a*) within the pons, by hemorrhage, tumor, or softening, with the production of crossed facial paralysis; (*b*) at its point of emergence, by tumors, meningitis, syphilis, or fractures at the base of the skull; (*c*) in the Fallopian canal, by diseases of the middle ear or by caries of the petrous portion of the temporal bone; (*d*) at its emergence

through the styloid foramen, by blows, cuts, tumors of the parotid gland, or by the pressure of the forceps in instrumental delivery; (*e*) exposure to cold is a frequent cause leading to a mild form of neuritis.

Symptoms.—In peripheral facial paralysis all the branches of the nerve are usually affected. The expression of the face is striking and characteristic. The paralyzed half of the face is lax, wrinkles are obliterated, and the naso-labial fold is no longer evident. The lower lid droops, the eye waters, and the eye cannot be closed voluntarily. The corner of the mouth sags, the mouth is drawn away from the affected side, and there is constant drooling of saliva. Mastication and articulation become impaired.

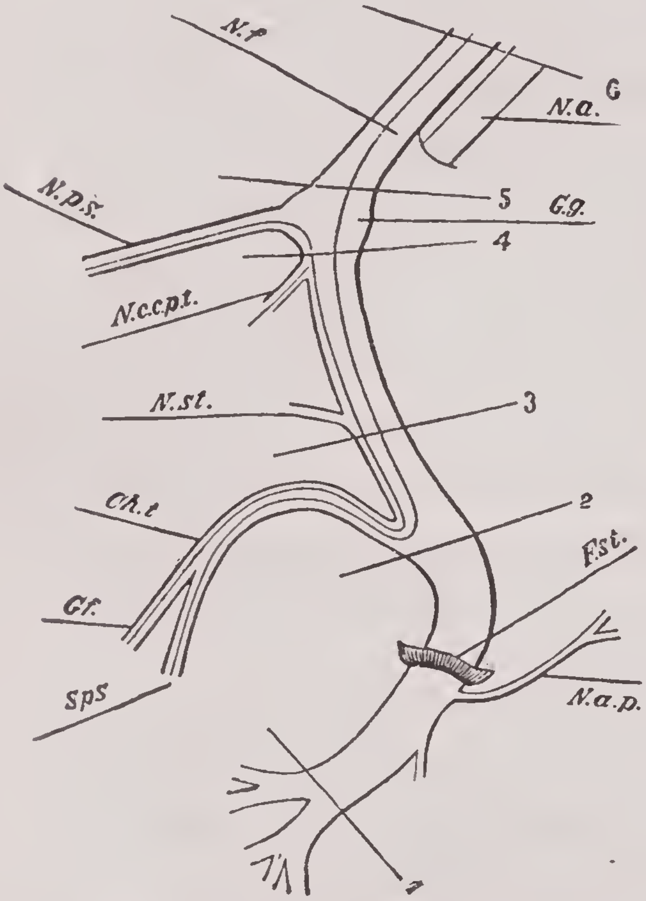

FIG. 63.—Schematic representation of the trunk of the facial from the base of the skull to the pes anserinus; different localizations of the lesion in paralysis (Strümpell): *N.f*, facial nerve; *N.p.s*, great superficial petrosal; *N.c.c.p.t*, nerve communicating with the tympanic plexus; *N.st*, stapedius; *Ch.t*, chorda tympani; *G.f.*, fibres of taste; *S.p.s*, nerve governing the secretion of saliva; *N.a*, acoustic nerve; *G.g*, geniculate ganglion; *F.st*, stylo-mastoid foramen; *N.a.p*, posterior auricular nerve.

In many cases there is said to be a paralysis of the soft palate, but this is doubted by Gowers. Facial paralysis is rendered evident by any attempt at talking, whistling, or inflating the cheeks. The exact localization of the lesion can readily be appreciated by an examination of the accompanying diagram (Fig. 63).

1. Paralysis of facial muscles; taste, secretion of saliva, and hearing are normal; the seat of the affection is in the portion between 1 and 2, usually in the trunk of the facial nerve, below the Fallopian canal.

2. Paralysis of the facial muscles, disturbance of taste, and eventually diminished secretion of saliva; hearing normal; the seat of the lesion is in the Fallopian canal, between 2 and 3.

3. Paralysis of the facial muscles, disturbance of taste, diminished secretion of saliva, abnormal acuteness of hearing; the seat of the lesion is between 3 and 4.

4. Paralysis of the facial muscles, disturbance of taste, diminished secretion of saliva, abnormal acuteness of hearing, and paresis of the soft palate (?); the seat of the lesion is in the geniculate ganglion, between 4 and 5.

5. Paralysis of the facial muscles, diminished secretion of saliva, abnormal acuteness of hearing, paresis of soft palate (?), but no disturbance of taste; the seat of the lesion is above the geniculate ganglion, between 5 and 6.

Erb distinguishes three grades of severity of facial paralysis:

1. *Mild Form.*—This form usually occurs after exposure to cold. The facial muscles are alone involved, there being no acuteness of hearing nor disturbance of taste. Electrical reactions remain good, and recovery follows after two or three weeks.

2. *Middle Form.*—There is a partial reaction of degeneration. The electrical excitability of the nerve is diminished, but not entirely lost. In two or three weeks the A. C. C. is found greater than the C. C. C., and the muscles react slowly to the galvanic current. Recovery ensues in from four to eight weeks.

3. *Severe Form.*—There is loss of faradic and galvanic excitability of the nerves; the galvanic excitability of the

muscles shows decided reaction of degeneration. Recovery does not occur before eight to fifteen months.

The **prognosis** of facial paralysis is usually good. In cases following traumatism, however, the damage done to the nerve may be permanent. In some cases, during convalescence, contractures are observed in the affected muscles, so that the wrinkles and folds of the skin may actually be deepened upon the affected side.

Spasm of the facial nerve is rare as the result of irritation of its nucleus. Usually spasm occurs as a habit in children or is due to reflex irritation. Tonic spasm may follow paralysis or may be due to catching cold. Facial spasm may be part of the affection known as "tic convulsif," characterized by spasms of various facial muscles and by explosive utterances, frequently of bad language.

AUDITORY NERVE.

The centre for hearing is in the first temporal convolution. Left-sided lesions in this location give rise to word-deafness. The fibres passing from the cortical centre to the auditory nucleus in the medulla may be destroyed by tumors of the corpora quadrigemina.

Nuclear degeneration is uncommon. The nerve itself may be compressed by meningitis or by tumors or fractures of the base of the skull. Neuritis may complicate locomotor ataxia, and is not uncommon with epidemic cerebrospinal meningitis, many cases of deaf-mutism following this latter disease. The labyrinthine branches may be involved by inflammation extending from the middle ear, by overdosing by quinine and the salicylates, and by the constant noises and jarring to which locomotive-makers and boilermakers are subject.

Destructive lesions in the course of the auditory tract give rise to "nervous deafness," frequently associated with tinnitus and vertigo. Sudden complete deafness is characteristic of syphilitic disease of the internal ear.

The **diagnosis** of nervous deafness from that produced by local disease of the middle ear is made by the use of the tuning-fork. If the vibrations of the tuning-fork are audible when the base of the instrument is placed against the temporal bone, nervous deafness may be excluded.

MÉNIÈRE'S DISEASE (Auditory Vertigo; Labyrinthine Vertigo).—At the present date all cases of aural vertigo are spoken of as instances of Ménière's disease, but more properly the name should be restricted to those cases with a sudden onset and recurring as a paroxysmal affection, originally described by Ménière in 1861.

Etiology.—The condition is most common in males after the fortieth year, and appears to bear no relation to middle-ear disease.

Pathology.—Whether the lesion be in the semicircular canals or in the cerebral centres is not known.

Symptoms.—The patient suddenly becomes giddy; surrounding objects seem to revolve, or the patient himself may seem to be gyrating, usually from the left to the right. The vertigo may render walking, or even standing, impossible. The onset may be so abrupt that the patient will fall, and may even lose consciousness for a short time. In a few minutes the dizziness passes away; the patient is left pale, prostrated, and bathed with a clammy sweat, and nausea and vomiting are apt to follow the attack. There is usually deafness in one ear, never complete.

The paroxysms recur at irregular intervals, and the prognosis is uncertain. Recovery occurs in some cases, while in others the attacks become more frequent and deafness may become complete.

Treatment.—Quinine in 20-grain doses daily for several weeks has been recommended by Charcot. Hypodermic injections of from 5 to 10 drops of a 2 per cent. solution of pilocarpine every second day are often of service, but the treatment should be restricted to robust patients. Nitroglycerin may be given if there be high arterial tension. Sodium bromide or hydrobromic acid is often of great service. Sodium salicylate in 5-grain doses three times daily is recommended by Gowers.

GLOSSO-PHARYNGEAL NERVE.

Nuclear degeneration gives rise to the pharyngeal symptoms of bulbar paralysis. The nerve-trunk may be involved by meningitis, or may be the seat of a diphtheritic neuritis, causing difficulty in swallowing and loss of sensation in the palate. Lesions of the root of the glosso-pharyngeal nerve do not give rise to loss of taste in the posterior third of the tongue, as the taste-fibres come originally from the fifth nerve.

PNEUMOGASTRIC NERVE.

The vagus gives branches to the pharynx, larynx, lungs, heart, œsophagus, and stomach, and is the chief afferent nerve of the vaso-motor centre. The nucleus may be involved by hemorrhage, tumor, softening, or by slow degeneration as in bulbar paralysis. The nerve-root within the cranium may be compressed by meningitis, by tumors, or by aneurysm of the vertebral artery. In the neck the vagus may be injured by wounds or may be accidentally cut or ligated in surgical operations. The recurrent laryngeal branches are frequently stretched by the growth of aortic or innominate aneurysms. Neuritis of the vagus is uncommon.

1. Pharyngeal Branches.—Motor branches from the pneumogastric combine with those from the glosso-pharyngeal to form the pharyngeal plexus. Paralysis may result

from bulbar degeneration or as the result of a post-diphtheritic neuritis. The symptom is difficulty in swallowing. Food may enter the larynx or may be regurgitated through the nose if the soft palate also be paralyzed.

Spasm is usually a neurosis, affecting nervous people. Its type is the "globus hystericus." Spasm is one of the principal symptoms of hydrophobia and of pseudo-rabies.

2. LARYNGEAL BRANCHES.—The superior laryngeal branch supplies the mucous membrane of the larynx above the vocal cords, and the crico-thyroid muscle. The recurrent laryngeal, arising in the upper part of the thorax, supplies the mucous membrane below the vocal cords, and all the muscles except the epiglottidean and the crico-thyroid. The motor fibres are derived originally from the spinal accessory.

The following chief forms of laryngeal paralysis are thus tabulated by Gowers:

Symptoms.	*Signs.*	*Lesion.*
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn together.	Total abductor palsy.
Symptoms inconclusive: little affection of cough or voice.	One cord near the middle line, not moving during inspiration; the other cord normal.	Unilateral abductor paralysis.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

The laryngeal palsies may be due to nuclear disease (as bulbar paralysis), to lesions of the trunk of the vagus or of the recurrent laryngeal nerves, to severe laryngeal catarrhs, or to over-use of the voice, or they may appear as hysterical conditions. For a more detailed description of the laryngeal palsies the reader is referred to specialized works upon the subject.

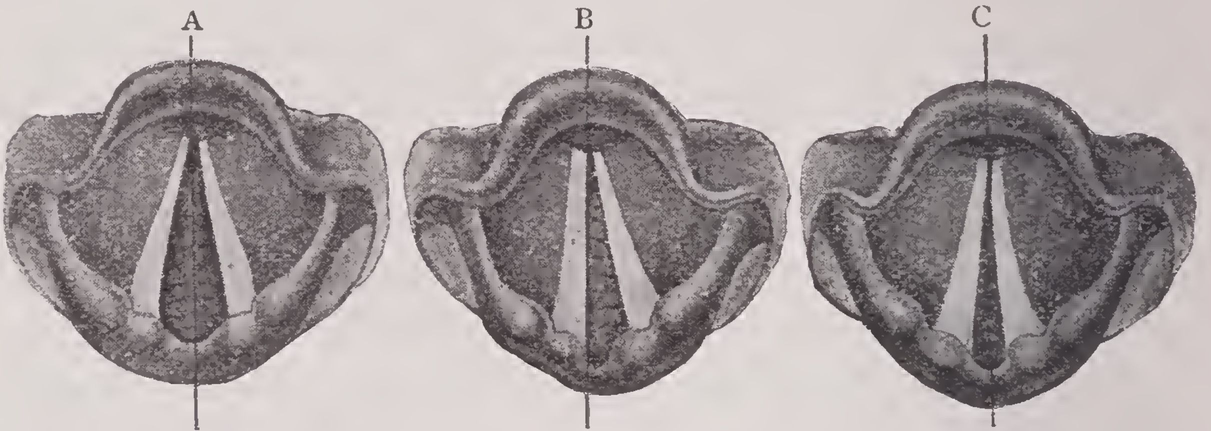

FIG. 64.—A, Adductor paralysis of both cords; B, adductor paralysis of left cord; C, abductor paralysis of both cords (Brown).

Spasm of the laryngeal muscles may occur in children (see Laryngismus Stridulus and Spasmodic Croup), or in adults as a laryngeal crisis of locomotor ataxia. Paroxysmal attacks of laryngeal spasm sometimes occur in adults, usually in the night; they have been known to replace migraine. Spasm may also be induced by irritation of the recurrent laryngeal nerve by the pressure on it of aneurysms or tumors, the spasmodic dyspnœa thus induced being relieved by whiffs of chloroform, while the obstructive dyspnœa of aneurysmal pressure on the trachea or of paralytic dyspnœa remains unchanged. Laryngeal spasm may occur with tetany or hysteria. A rare condition of functional spasm occurs when attempts are made to speak, the cords being brought together too forcibly.

3. PULMONARY BRANCHES.—The spasmodic contraction of the muscular fibres of the bronchial walls in asthma is thought to be induced by a neurosis of the vagus. Destructive lesions of the nucleus of the pneumogastric, as hemorrhage or softening, are followed by rapid congestion of the lung, with extravasation of blood into the pulmonary tissue.

4. Cardiac branches are motor, sensory, and trophic in character.

(*a*) *Motor.*—Irritation of the vagus causes an inhibitory slowness of the heart's action. Bradycardia may thus follow irritation of the vagus-nucleus or compression of the nerve in the neck by tumors, or the slowing of the pulse may be an evidence of a pure neurosis of the nerve. Paralysis of the vagus abolishes inhibitory action and allows the accelerators full sway. Tachycardia may thus occur with diphtheritic neuritis, wounds or accidental ligature of the vagus, or the involvement of the nerve-trunk by tumors or by toxæmic conditions depressing the activity of the vagus-nucleus.

(*b*) *Sensory.*—The vagus is the afferent nerve of the heart, and through it the unpleasant sensations of palpitation and pain are conveyed to the brain. The relation of the pneumogastric nerve to angina pectoris is not thoroughly understood.

(*c*) *Trophic.*—After injury of the vagus the heart is usually found in the condition of acute fatty degeneration.

5. Œsophageal Branches.—Paralysis of the œsophagus gives rise to difficulty in swallowing, simulating stricture. Spasm of the œsophagus is more frequent than paralysis.

6. Gastric branches are sensory and motor.

(*a*) *Sensory branches* may be the seat of pain, either from irritation of the terminal fibres or from spontaneous neuralgia. Hunger is generally believed to be a pneumogastric sensation, and it is probable that many cases of nervous dyspepsia are dependent upon deranged functional activity of the vagus nerve.

(*b*) *Motor.*—After section of the nerve, contraction-power of the stomach is lessened but not altogether lost. The cerebral vomiting of meningitis and of other brain diseases is due to irritation of the vagus-nucleus, and paroxysmal vomiting may be due to an intermitting pressure on the pneumogastric nerve in the neck.

7. INTESTINAL BRANCHES accelerate the action of the intestines, but intestinal symptoms from diseases of the nerve or of its nucleus are exceedingly rare.

SPINAL ACCESSORY NERVE.

The "accessory" portion, arising from the medulla, joins the pneumogastric and supplies the laryngeal muscles. The "spinal" portion, arising from the cervical portion of the cord, supplies the sterno-mastoid and the portion of the trapezius muscle between the occipital bone and the acromion. The nucleus in the medulla may be involved in bulbar paralysis; the nerve-trunk may be implicated by meningitis, tumors, or caries. The symptoms of nuclear disease are those of the various laryngeal palsies. Disease of the spinal portion is followed by paralysis of the sterno-mastoid and of the upper portion of the trapezius. There is an absence of the normal prominence of the sterno-mastoid muscle in the neck, and the head is rotated with difficulty to the opposite side. The head may be held obliquely, but purely paralytic torticollis does not occur. The paralysis of the upper portion of the trapezius gives a concave contour to the neck, especially marked on deep inspiration; the shoulder falls a little, the scapula recedes from the spine, and the angle is rotated inward from the unopposed action of the rhomboids and the levator anguli scapulæ. Elevation of the arm is also impaired, as the deltoid loses some of the support from which it acts. Bilateral paralysis may occur with progressive muscular atrophy, and is not uncommon in children in consequence of chronic meningitis about the foramen magnum damaging both spinal accessory nerves. If both sterno-mastoids are affected, the head falls backward; if both trapezii, the chin sinks upon the sternum.

SPASM OF THE SPINAL ACCESSORY NERVE (Torticollis; Wry-neck).—1. The congenital form (fixed wry-neck) affects the right side almost exclusively. The sterno-mastoid is shortened, frequently is atrophied, and usually is hard and firm. In some of the cases the condition is thought to depend upon injury to the muscle from traction upon the neck during birth. The muscle stands out prominently; the head deviates and cannot be turned toward the side on which the muscle is contracted. The symptoms are rarely noticed during early childhood, because of the shortness of the neck. In many cases there is an associated facial asymmetry, which by some is thought to be an essential feature of the congenital form. Congenital torticollis is of slight importance, as it can readily be cured by tenotomy.

2. SPASMODIC TORTICOLLIS may be either clonic or tonic, the two varieties being frequently combined. The condition seems to be more frequent in males, usually between thirty and fifty years of age; most cases occurring in females under the age of thirty are of a hysterical nature. There is frequently a neurotic family history, but in the majority of cases no exciting cause can be demonstrated.

Symptoms.—Spasm may be the first symptom, but in many cases there is a preceding pain of a sharp neuralgic or a dull character, or a sense of stiffness may antedate the spasm. The spasm usually comes on gradually, and may involve the sterno-mastoid alone, or there may be an associated spasm of the upper portion of the trapezius on the same side, or of the splenius, usually upon the opposite side. The scaleni and platysma myoides occasionally contract, or more rarely the deep cervical muscles. Bilateral contraction of the sterno-mastoid muscles causes a backward movement of the head, so that the face may look upward (retrocollic spasm), and there is an associated spasm of the frontales muscles. The various other deformities may be inferred by a knowledge of the anatomy of the affected muscles. Clonic spasms may recur every few minutes, are usually associated with considerable pain, and in time the muscles become hypertrophied. The clonic spasms cease during sleep and are aggravated by emotion, excitement, or fatigue. In some cases the spasms spread to the muscles of mastication, of the face, or of the arms.

Prognosis.—The course of the disease varies. After a few years the disease ceases to progress and may either remain stationary or improve. Recurrences are common. The disease is usually regarded as a functional neurosis.

Treatment.—The spasm may be relieved by valerianate of zinc, by asafœtida, or by sodium bromide. Chloral and cannabis indica are frequently of service. Hypodermic injections of morphine constitute the most curative mode of treatment, but from the long continuance of the treatment the habit is regularly formed, so that the treatment should not be inaugurated. Galvanism should be tried. In obstinate cases nerve-stretching, division, and excision may be performed.

HYPOGLOSSAL NERVE.

Nuclear disease is usually degenerative in character, forms part of bulbar paralysis, and may occur with locomotor ataxia or from acute softening from obstruction of the blood-vessels. The symptoms are usually bilateral.

Supranuclear disease may occur anywhere in the motor tract between the nucleus and the lowest portion of the ascending frontal convolution. Paralysis of the tongue occurs upon the opposite side, and is usually associated with hemiplegia. In supranuclear disease the tongue does not waste.

In infranuclear disease the fibres of the nerve may be involved by tumor or meningitis, or the nerve may be compressed within its foramen by caries of the bones. There may be a neuritis of rheumatic or saturnine origin. In nuclear and infranuclear disease the tongue wastes and the reaction of degeneration is present.

Symptoms.—In unilateral paralysis the tongue, when protruded, deviates to the paralyzed side; in bilateral palsy it lies motionless and cannot be protruded. Articulation and mastication are interfered with in proportion to the extent of the paralysis. Sensation and taste are not impaired. If the hypoglossal fibres be involved within the nucleus or after leaving the nucleus, there may be paralysis of the tongue on the same side, with opposite hemiplegia (Fig. 57).

Spasm of the tongue may be part of a general spasm, such as epilepsy or chorea; the affection occurs in some forms of stuttering. The tongue is a not infrequent seat of spasm in hysteria. Spasm of the tongue may also be induced by reflex irritation of the fifth nerve.

A rare form of paroxysmal clonic spasm is described, in which the tongue is protruded as many as forty or fifty times a minute. Spasm of the tongue is almost regularly dependent upon functional states of the nervous system that are removable by tonic treatment.

6. DISEASES OF THE PERIPHERAL NERVES.

NEURITIS.

Neuritis may be localized in one nerve or may involve a number of nerves. For convenience, therefore, a localized and a general or multiple form are described.

Localized Neuritis.

Etiology.—Traumatism is the most frequent cause for a localized neuritis, the nerve being injured by wounds, contusions, fractures and dislocations of bones, sudden violent muscular exertion, by hypodermic injections of irritants such as ether, and by the steady strain on certain muscles, as in professional palsies. The nerve may be involved by an extension of inflammation from other parts, especially from bone disease. A nerve-trunk may also be involved by the growth of tumors. Cold is a not infrequent cause of neuritis, the facial and sciatic nerves being most commonly affected from this cause. This form is often spoken of as "rheumatic neuritis." The various toxic agents to be considered under Multiple Neuritis may act upon a single nerve.

Pathology.—An interstitial and a parenchymatous neuritis occur.

Interstitial Neuritis.—The inflammation may involve the perineurium or may extend into the deeper portions. The connective tissue becomes congested and infiltrated by leucocytes. The nuclei of the sheath of Schwann are increased, and the myelin becomes broken up into fragments. The axis-cylinder may ultimately develop degenerative atrophy, so that in the final stages the nerve is represented by a strand of very fatty connective tissue (the "lipomatous neuritis" of Leyden). Interstitial neuritis is the form which usually occurs from extension of inflammation from carious bone.

Parenchymatous Neuritis.—Congestion and exudation occur in the nerve-trunk, and there is a degeneration of individual nerve-fibres. The myelin-sheath becomes disintegrated into fatty granules; the nuclei of the sheath of Schwann multiply. In severe cases the axis-cylinder also undergoes fatty disintegration. The products of degeneration liquefy and are absorbed, leaving only the collapsed sheath of Schwann. In this form, which resembles the secondary degeneration occurring in a nerve divided from its trophic centre, the interstitial connective tissue is but slightly affected. The muscles supplied by the degenerated nerve undergo marked atrophic changes. This form occurs in traumatic cases. Regeneration occurs by the growth of new fibres outward into the affected districts of the nerve.

The two forms are frequently combined in non-traumatic cases.

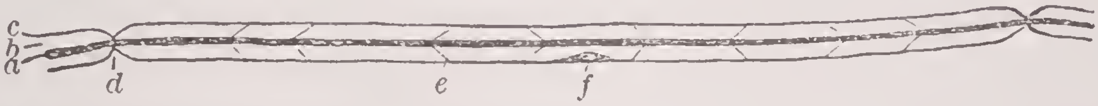

Fig. 65.—Normal nerve-fibre (Starr): *a*, axis-cylinder; *b*, medullary sheath; *c*, sheath of Schwann; *d*, node of Ranvier; *e*, incisure of Schmidt; *f*, nucleus of sheath of Schwann.

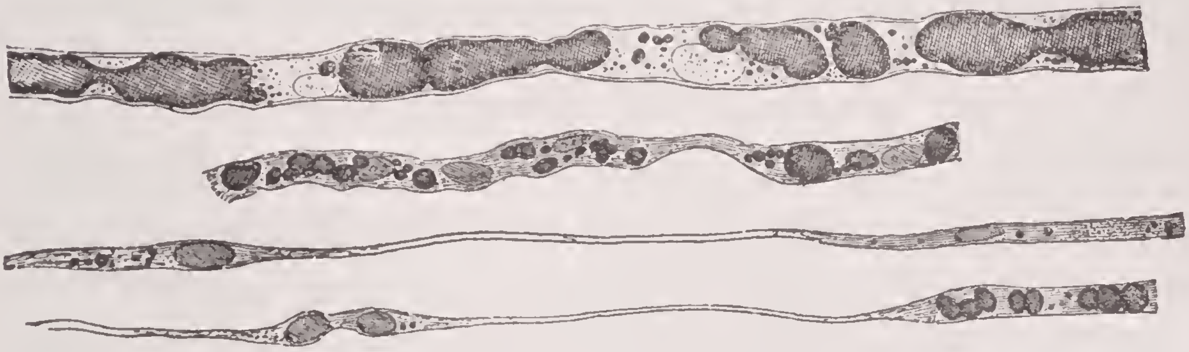

Fig. 66.—Degeneration of nerve-fibres in various stages (Starr).

Symptoms.—There is weakness or paralysis of the muscles to which the nerve-fibres are distributed; motion is painful, and there may be at the onset twitchings or contractions. Atrophy of the muscles ultimately develops; there is loss of the faradic irritability, and the reaction of degeneration develops. Pain, of a boring or stabbing character, is felt along the course of the nerve and in its area of distribution, followed by numbness or anæsthesia. Loss of tactile sensation frequently occurs even if the pain be marked. Tenderness along the course of the nerve-trunk usually persists. Trophic changes may become evident: the skin becomes glossy or reddened and œdematous; the nails become defective; and there may be herpes, increased surface temperature, localized sweatings, and effusion into the joints. A localized neuritis may in rare instances extend upward along the larger nerve-trunks (ascending or migrative neuritis), and may even reach the spinal cord, causing a subacute myelitis.

Prognosis.—The course of the disease is usually slow, but recovery should occur if the continuity of the nerve-fibre be preserved. Mild cases of traumatic neuritis may pass away within a few days.

Treatment.—If the nerve be cut, the divided ends should be sutured on well-known surgical principles. The injured part should be placed at rest in a padded splint, or hot applications may first be applied for the pain. Electricity and massage are of service to the paralyzed muscles.

MULTIPLE NEURITIS.

Etiology.—The causes of multiple neuritis may be thus tabulated: (1) The *toxines of infectious diseases*, as diphtheria, typhoid fever, small-pox, leprosy, scarlet fever, syphilis, tubercle, grippe, malaria, and beri-beri; (2) *chemical poisons*—ether, alcohol, coal-gas, bisulphide of carbon, naphtha, lead, arsenic, mercury, phosphorus, copper, zinc, ergot, and morphine; (3) *auto-toxines* with rheumatism, gout, arthritis, diabetes, the puerperal state, and chorea; (4) *cachectic conditions*, such as anæmic, cancerous, or tuberculous cachexia; (5) *idiopathic* (?) *cases* following exposure to cold or over-exertion.

Pathology.—The lesions may be of an interstitial neuritis alone, of a parenchymatous neuritis alone, or the two forms may be combined. The peripheral parts of the nerves are more seriously affected than are the central parts. The neuritis may involve motor nerves alone (as in lead-poisoning), or sensory nerves alone (as in coal-gas poisoning) or both sensory and motor nerves may be affected (as in alcoholic neuritis).

Symptoms.—The symptoms are those of neuritis, previously described, differing only from those of the localized form in being more general in their distribution. As the symptomatology of multiple neuritis is so varied, it will be more convenient to describe separately the principal clinical types.

Acute Febrile Polyneuritis.—The onset is acute, with a chill, fever of from 103° to 104° F., and pains in the back and the limbs. Pain and tingling occur in the peripheral parts, and the nerve-trunks are exquisitely tender on pressure. Motor weakness becomes evident in a few days, the extensors being the more seriously involved. The paralysis rapidly extends up the extremities, and may even involve the trunk or the face. The muscles rapidly waste, faradic irritability is lost, and the reaction of degeneration appears. Trophic changes are commonly observed. The clinical picture is that of an acute ascending palsy, resembling or, according to some, being identical with Landry's paralysis. Death occurs in from one to three weeks in the severe cases. In milder cases, after the symptoms have persisted for from four to six weeks slow improvement begins, but recovery rarely is complete under one or two years. In some cases the onset is more gradual and the course of the disease more prolonged, so that the clinical type resembles that of Duchenne's paralysis.

Alcoholic neuritis, the most common form of the disease, occurs more frequently in women than in men. The onset may be acute, accompanied by a chill and by fever of from 101° to 103° F.; or the disease may begin gradually, with sensory symptoms in the feet and the hands, such as neuralgic pains, tingling and pricking feelings, and cramps in the muscles. There is considerable tenderness along the course of the nerve-trunks. Paralysis next occurs, at first in the feet, then in the hands and forearms, and the characteristic wrist-drop and foot-drop are developed (Fig. 67). The affected muscles undergo rapid atrophy and show the reaction of degeneration. The superficial and deep reflexes are lost. Sensory symptoms may consist of numbness or tingling, or there may be severe neuralgic pains. There may be areas of anæsthesia and of retarded sensation. The combination of anæsthesia of the skin with extreme hyperæsthesia and soreness of the muscles is highly suggestive of the alcoholic form of neuritis. The paralysis may remain localized in the hands and feet, and after a stationary period of several months slow spontaneous recovery will ensue, the duration of the disease being from six to twelve months. As the extensors of the feet usually remain paralyzed for some time, the "steppage gait" becomes prominent and is characteristic of peripheral neuritis. In other and more malignant cases there may be spreading paralysis of the extremities, trunk, sphincters, and even of the face, and death may be caused by involvement of the muscles of respiration.

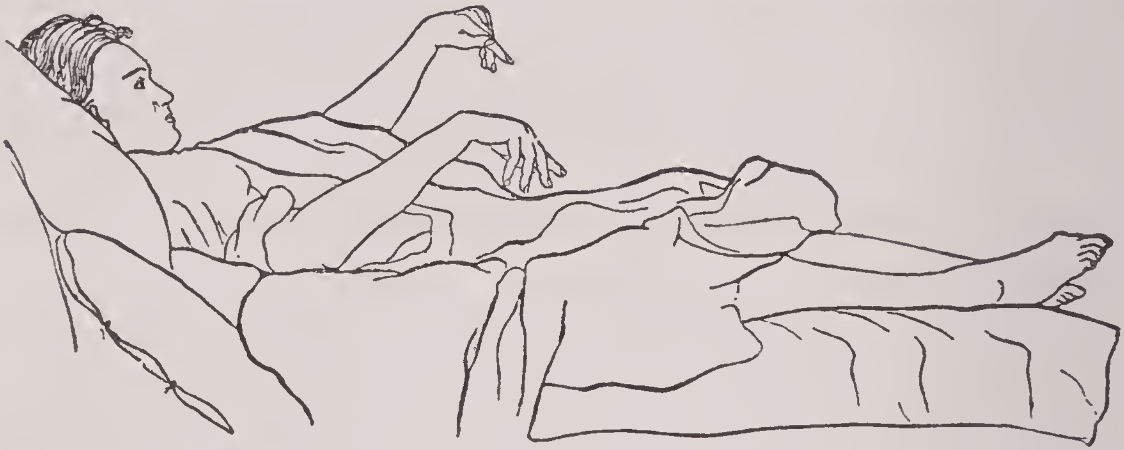

FIG. 67.—Multiple alcoholic neuritis: wrist-drop and foot-drop (Gowers).

The cerebral symptoms of alcoholic neuritis are frequently well marked. There may be convulsions at the onset or throughout the disease. Delirium with extravagant hallucinations is usually well marked, and may verge into the type of delirium tremens. Appreciation of time and place is usually lost. In some cases the patient passes into the typhoid condition, with low, continued fever and muttering delirium.

Post-febrile Neuritis.—The various forms of post-febrile neuritis have been described under the respective diseases that precede them. Diphtheritic paralysis, the most common and serious form, may involve the muscles of the palate, eye, or heart, or may be generally distributed in the extremities. There are no sensory symptoms in the majority of cases of neuritis following other infectious diseases, and the distribution of the paralysis is usually paraplegic.

Lead-paralysis is usually preceded by anæmia, colic, constipation, and the blue-black line on the margin of the gums. The symptoms of lead-palsy may appear abruptly or gradually, and are not attended by sensory disturbances. The following localized lead-palsies are described:

1. Anti-brachial type—paralysis of the extensors of the fingers and the wrists. The musculo-spiral nerve is involved, causing the characteristic wrist-drop.

2. Brachial type, involving the deltoid, biceps, brachialis anticus, and the supinator longus, more rarely the pectorals. This form is bilateral, and may follow the first type or be primary.

3. Aran-Duchenne type, involving the small muscles of the hands, closely resembling the earlier stages of progressive muscular atrophy.

4. Peroneal type, involving the lateral peroneal muscles and the extensors of the toes, producing the "steppage gait."

5. Laryngeal type, involving the adductors of the larynx.

In other cases the paralysis becomes more generalized, gradually involving the extremities. There are occasionally seen acute cases resembling Landry's paralysis. There has been described a rare form of lead-neuritis in which atrophy and paralysis come on together and develop proportionately.

The prognosis of lead-palsy is generally good. The course of mild cases is about four months.

Arsenical paralysis is rare. The symptoms are chiefly motor, resembling those of alcoholic neuritis. The "steppage gait" is usually well marked. Recovery usually occurs in from two to six months.

Coal-gas neuritis is generally slight, and involves only sensory nerves. Numbness persists for a long time in the hands and feet.

BERI-BERI, OR KAK-KE.—This disease, which occurs endemically in Northern Brazil, China, Japan, India, the Straits Settlements, and in the Malay Archipelago, is a multiple neuritis probably due to infection by a micrococcus. Foreigners in the endemic localities are usually exempt. At times the disease assumes epidemic proportions; in 1878, 38 per cent. of the Japanese army were affected. Nothing is accurately known as to the etiology.

Symptoms.—Two principal forms are described—an œdematous or "wet" and a paralytic or "dry" form. The œdematous form begins acutely, with fever, œdema, paresis, and numbness of the lower extremities. Effusion into serous cavities may occur. The liver and spleen are enlarged. The dry form may occur primarily or may be a development of the wet variety. Sensory symptoms, which are prominent, consist of anæsthesia and prickling and tingling sensations. Paresis, atrophy, and the "steppage gait" develop. A malignant form of the dry variety occasionally occurs, characterized by rapidly ascending paralysis and by suppression of urine.

The **prognosis** of the mild forms is good, but relapses are common. In some epidemics, however, the mortality is over 40 per cent.

Treatment of Multiple Neuritis.

Rest in bed is essential, and salicylic acid or the salicylates are recommended during the earlier stages of acute cases with fever. Warm applications to the affected limbs should be made by packs or baths. After the acute stage has passed, gentle rubbing of the muscles with oil and massage are to be employed. Warm baths of 98° F. are to be given for half an hour several times a day. Contractures are to be overcome by passive movements and by extension. The interrupted galvanic current should be employed daily. Alcohol should be cut off from all cases, although in the alcoholic form the reduction should be gradual. Of drugs during the chronic stage, arsenic and strychnine are the most valuable. The strength should be supported by careful regulation of the diet, and the use of cod-liver oil is generally advisable.

NEUROMATA.

Tumors situated in the nerve-trunks may be composed of nerve-tissue (the true neuromata) or of other tissues (the false neuromata).

False neuromata are chiefly formed of fibrous tissue. More rarely there occurs myxomatous tissue, sarcoma, or an infiltration of the nerve-fibres by carcinoma. In leprosy the nerves are frequently much thickened by an infiltrating growth of connective tissue. A curious variety is the *plexiform neuroma*, which consists of interlacing cords more or less nodular. In this condition, which is usually of congenital origin, hundreds of fibrous nodules may occur along the course of the nerve-trunks. The symptoms of false neuromata are seldom evident, although in some cases the symptoms of neuritis may appear.

True neuromata, containing nerve-fibres, rarely ganglionic cells, may form small subcutaneous painful tumors—tubercula dolorosa. These tumors are not always, however, pure neuromata, but may consist of fibrous tissue or may be adenomatous growths of the sweat-glands. True neuromata may develop on the cut ends of nerves in amputation-stumps, in which situation they may lead to great pain and distress.

Treatment.—When painful, the growths may be excised.

NEURALGIA.

Etiology.—Under the term "neuralgia" are included painful affections of the nerves due either to functional disturbance or to neuritis. The causes and symptoms of neuritis have elsewhere been described.

Members of neurotic families are subject to neuralgic affections. Women are more often affected than are men, but children are usually exempt.

General debility, anæmia, excesses, over-work, and nervous exhaustion are potent predisposing factors. Neuralgia is not uncommon with malaria, rheumatism, gout, syphilis, diabetes, and chronic lead-poisoning. Reflex irritation may act as an exciting cause; thus obstinate trifacial neuralgia may result from carious teeth or from eye-strain. In susceptible patients exposure to wet and cold may induce an attack.

Pathology.—Aside from the cases in which neuralgia is dependent upon neuritis, the disease is a pure neurosis.

Symptoms.—The attack may be preceded by chilliness, mental depression, or tingling in the part to be affected. The chief symptom is pain, of a paroxysmal, stabbing, burning, or darting character, localized in certain groups of nerves. The skin of the affected area may be hyperæsthetic, and spots of exquisite tenderness are detected at certain points along the nerve where it makes its exit through a bony canal or a fibrous sheath. Vaso-motor symptoms may accompany the pain; the skin may be cool, or hot and burning, and there may be areas of œdema or of erythema or herpes. Twitching of the muscles, or even spasms, may occur during the paroxysm. The attack lasts from a few minutes to a number of hours and then subsides. The interval between the paroxysms varies in different cases. Frequently the recurrences are at regular intervals, often only at the menstrual period.

Clinical Varieties.—*Trifacial Neuralgia* (Tic Douloureux).—The pain is felt in one or more branches of the nerve, the ophthalmic division being most frequently affected. Hyperæsthesia of the skin and of the mucous membranes is common, and vaso-motor phenomena are not infrequently present—flushing, sweating, salivation, increased nasal discharge, and lachrymation. There may be trophic changes—erythema, induration of the skin, loss of hair or local grayness. In severe cases there may be an associated spasm of the facial muscles—the "tic convulsif." Tender points correspond to the supraorbital, infraorbital, and mental foramina, less frequently to the occipital protuberance and the upper cervical spine. Trifacial neuralgia is frequently of reflex origin.

Cervico-occipital Neuralgia.—The pain, which is usually dull and more or less constant, is localized over the back of the neck and the head, extending forward as far as the parietal eminences and the ear. There is frequently hyperæsthesia of the scalp. The most important tender point is located midway between the mastoid process and the spine, where the great occipital nerve becomes superficial. Exposure to cold and cervical caries are the most frequent causes of this form of neuralgia.

Cervico-brachial neuralgia, which involves the sensory nerves of the brachial plexus, is usually most intense in the axilla or along the course of the ulnar nerve. When the circumflex nerve is involved the pain is in the deltoid. The pain may be so increased by movement as to render the arm helpless. The most common tender points are the axillary, the circumflex at the posterior border of the deltoid, the superior ulnar behind the elbow, and the inferior ulnar in front of the wrist. Cervico-brachial neuralgia more frequently than any other form is the result of injury. Some severe forms are evidences of an occupation-neurosis.

Intercostal neuralgia is very common in hysterical and anæmic women. Pain is felt along the intercostal nerves in aneurysm, caries, and pleurisy. There is usually a dull, constant pain, with acute stabbing exacerbations. Painful points are detected beside the vertebra, under the angle of the scapula, and under the breast. Pleurodynia differs from true intercostal neuralgia in being localized in one spot not corresponding with the course or exit of the intercostal nerves. The pain is increased by expansion of the thorax rather than by lateral movements of the trunk. Herpes zoster occurs with the most aggravated form of intercostal neuralgia, which may persist after the eruption has subsided. The eruption and the neuralgia are due to neuritis.

Lumbar neuralgia gives rise to pain along the crest of the ilium, the inguinal canal, and the spermatic cord, and in the testis, scrotum, and labium majus. Irritable testis is usually accompanied by syncopal sensations.

Coccygodynia, which is common in women, is aggravated by the sitting posture. This form of neuralgia is usually very intractable.

Sciatica.—The pain extends down the back of the thigh, often reaching as far as the foot. The pain may be uniformly distributed along the course of the nerve, but not infrequently there are spots in which it is more intense. The pain is usually more or less constant and of a gnawing, burning character, but it may be paroxysmal, the paroxysms being usually more intense in damp weather and at night. The pain is regularly increased by walking; the knee is bent and the patient walks on the toes to diminish the tension on the nerve. The painful points are located (1) above the hip-joint, near the posterior iliac spine, (2) at the sciatic notch, (3) about the middle of the thigh, (4) behind the knee, (5) below the head of the fibula, (6) behind the external malleolus, and (7) on the back of the foot. Tenderness is usually also elicited by pressure along the course of the nerve. Muscular wasting and fibrillary twitchings complicate the severe cases.

Sciatica is most common in those with a gouty or rheumatic tendency. The nerve may be compressed by intrapelvic growths or may be involved by spinal caries.

The **prognosis** must be made with caution, as many cases of neuralgia prove intractable to treatment.

Treatment.—All causes for reflex irritation must be discovered and removed if possible. A tonic and supporting treatment is of the greatest importance. The patient should be built up in every possible way. Iron and arsenic are required for anæmic conditions; gouty and rheumatic taints are to be treated; quinine is to be given to malarial patients. The diet should be generous. Fats are indicated in nearly all of the cases, and a liberal amount of meat is to be allowed to all except those subject to gout. Many obstinate cases are benefited by residence in a dry inland climate. Strychnine, phosphorus, and cod-liver oil are of great service.

For the pain, antipyrine, phenacetine, lactophenin, chloral, croton-chloral, the bromides, and cannabis indica may be given. Aconite and gelsemium are recommended for trifacial neuralgia. Morphine, codeia, and hypodermic injections of cocaine are to be withheld, because of the danger of forming the habit. Local applications are frequently of service. Heat, stimulating liniments, freezing of the skin by ether or methyl-chloride sprays, blisters, or application of the actual cautery may be employed. Surgical treatment may be required for obstinate cases. Nerve-stretching is not likely to be followed by permanent results. Neurectomy, or the excision of a portion of the affected nerve, is frequently followed by good results, but the pain may return in time.

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7. GENERAL NERVOUS DISEASES.

INFANTILE CONVULSIONS; INFANTILE ECLAMPSIA.

Etiology.—Convulsions are so frequent in children that a special mention is justifiable. Owing to the lack of development of the higher cerebral centres of children, the lower centres are but improperly controlled, so that increased reaction to direct or reflex stimulation is permitted. The most important causes for convulsive seizures in children are the following:

1. *Rickets.*—Convulsions, usually without marked febrile disturbance, occur from slight causes, and are apt to be repeated at intervals for months.

2. *Gastro-intestinal Irritation.*—This most common cause arises from dietetic errors, indigestion, or worms. The convulsions are usually accompanied by fever.

3. *General exhaustion*, especially if due to diarrhœal disease. Convulsions may be part of a hydro-encephaloid condition.

4. *Mechanical congestion of the brain*, as with violent attacks of coughing.

5. *Deficient aëration of blood*, as with croup, diphtheria, or vitiated air in incubators.

6. During the first few days of life, from *severe brain-injury during birth.* If the convulsions be severe and persistent, meningeal hemorrhage should be suspected.

7. *Peripheral irritation*, as teething, phimosis, or otitis.

8. *Acute febrile conditions*, especially at the onset of measles, scarlet fever, and pneumonia.

9. Convulsions may usher in or accompany any serious *disease of the nervous system* in children.

10. Convulsions in infancy are not infrequently *epileptic*.

The **symptoms** may be preceded by signs of irritation of the nervous system—restlessness, irritability, and twitchings. The attack begins with a fixation of the eyeballs; the face becomes pale, the limbs and trunk become rigid and stiff. The fingers and toes are inverted (carpopedal spasm). Respiratory movement is impaired, so that the face becomes cyanotic. The spasm may relax, or may become clonic as in epilepsy. In some cases clonic spasms are marked from the start, and usually begin in the hands and face.

The spasm may be followed by muscular rigidity for some little time. In convulsions due to indigestion the attack may be single; in other cases attacks follow each other with great rapidity. Attacks coming irregularly and without assignable cause in children over two years of age are likely to prove to be true epilepsy. Convulsions may be followed by slight paresis or may lead to meningeal hemorrhage with hemiplegia.

The **prognosis** is usually good. A dubious prognosis should be given, however, in the case of weakly subjects, as fatal exhaustion may be induced.

Treatment.—The first and most important measure is to search for the cause of the seizure, and to remove it if possible. If indigestion be the cause, a prompt emetic should at once be given, or the stomach may be washed out. For the attack itself, if severe, whiffs of chloroform should be given, and an enema containing chloral (gr. ij) and sodium bromide (gr. v-x) should be administered, these doses being suitable for a child of from six to twelve months. No time should be lost in immersing the child in a bath at 95° F.; baths of a higher temperature are not suitable. After the bath an ice-cap should be employed or cold applications should be made to the head. Morphine may be necessary in case of recurring convulsions, but the drug should be administered with extreme caution, and should never be ordered for infants under six months of age. For a child of one year, gr. $\frac{1}{100}$ to $\frac{1}{75}$ hypodermically will be a sufficient dose.

EPILEPSY.

Etiology.—Among the remote causes which induce this disease, heredity is the most important, a neurotic family history being obtained in about one-quarter of all cases. A direct inheritance of epilepsy is rare, but the parents are apt to suffer from nervous diseases or to be the victims of the alcohol habit. Any vicious influence deteriorating the parent stock predisposes to the development of epilepsy in the offspring. Consanguineous marriages exert a distinct influence upon the causation of the disease. Epilepsy may interchange with insanity in different generations.

An immediate exciting cause is determined in but one-third of all cases. There may be blows on the head, dissipation, fright, or continued reflex irritation. Many cases in children date from teething or from acute infectious diseases. Although in many cases the exciting cause may be removed, the nerve-centres may have formed the habit of discharging nerve-force, so that the seizures continue through life.

Males are more frequently affected than females. The disease appears before the thirteenth year in one-third of the cases, between the thirteenth and nineteenth years in one-third, and between the nineteenth and thirtieth years in one-third of the cases. After the thirtieth year idiopathic epilepsy is rare. "An epilepsy which develops after the thirty-fifth year of age is not idiopathic, but is due to some organic brain disease, to the abuse of alcohol, to reflex irritation, or other causes, which in some cases may be so hidden as to be exceedingly difficult of recognition" (H. C. Wood). In the great majority of cases, recurring epileptic seizures in those over the age of thirty are due to brain syphilis.

The **pathology** of epilepsy is obscure. A degeneration of the neuroglia in the brain has been described by some observers. The generally accepted theory is that the seizures are due to a discharging lesion of the brain-cortex, so that an overflow of nerve-force occurs.

Symptoms.—Loss of consciousness with general convulsions is known as *grand mal;* loss of consciousness without convulsions, as *petit mal.* Localized convulsions, usually without loss of consciousness, are described as *Jacksonian* or *cortical epilepsy.*

GRAND MAL.—The attack may be preceded by peculiar sensations, described as *auræ,* which give warning to the patient that an attack is impending. Sensory auræ are the most common; they differ in character in different patients, but are constant in the one subject. An aura consists of a peculiar sensation, felt in some part of the body, mounting upward to the head. Auræ of special senses are occasionally observed. An ocular aura consists of visual sensations—flashes of light or of color, bizarre forms, double vision, or even blindness. In auditory auræ abnormal sounds or voices may be heard. An olfactory aura, which takes the form of a bad smell, is rather infrequent. Psychical auræ are not uncommon; the patient may feel alarmed or may be in terror, or there may be a vague sense of strangeness or a dreamy sensation. In some cases the attack is preceded by forced movements; the patient may run forward with great speed ("procursive epilepsy"), or may turn rapidly around as if on a pivot. In many cases the aura is absent.

The fit is usually abrupt; the patient falls to the ground unconscious. Preceding the attack there is generally a wild, harsh scream or groan. The fit occurs in three stages:

1. *Tonic Spasm.*—The head, eyes, and mouth are rotated to the side on which the spasm is more intense; the body is stiff and rigid. The hands are clenched, the arms and forearms flexed, the legs extended, and the feet extended and inverted. The body, however, is not always distorted in the same manner, as the muscular spasms may not be equally intense. The face, at first pale, becomes dusky or livid, owing to the respiratory spasm. The tonic stage lasts for from a few seconds to one or two minutes.

2. *Clonic Stage.*—Tremulous vibrations occur in the muscles; the vibrations increase in range until the limbs are jerked and tossed violently about. The face is frightfully contorted, foamy saliva is forced from the mouth, and the tongue is apt to be severely bitten. Respirations are noisy and stertorous. Urine and feces may be passed involuntarily, especially in nocturnal attacks. The pupils are immovably dilated, and after the attack usually show remarkable oscillations. The temperature is usually normal, but in case the attack be prolonged a slight rise, rarely to 102° F., may be observed. The clonic stage lasts for three or four minutes, and the patient passes into the—

3. *Stage of Coma.*—The patient becomes quiet and passes into a deep sleep, awakening after a few minutes or hours with headache, mental confusion, and muscular soreness. After the attack the reflexes are usually increased. The urine is usually increased in quantity after the attack, and may contain albumin. In rare instances the patient passes from one spasm into another without regaining conscious-

ness. In this *status epilepticus* the temperature may rise to 107° F., and the patient is apt to die from exhaustion.

Post-epileptic Symptoms.—The patient may emerge from the coma in a peculiar trance-like condition, and may perform purposeless and incongruous actions, at times so apparently rational that it may be impossible to believe that the patient is not conscious. At times this condition of *epileptic automatism* passes into the condition of epileptic mania, in which condition the patient is dangerous or even homicidal. After the attack slight transient hemiplegia or aphasia may be noticed. Epilepsy is frequently succeeded by mental degradation which may ultimately lead to complete dementia.

Nocturnal epilepsy may occur without the patient's knowledge, so that the condition may exist for years before the diagnosis is suspected.

2. PETIT MAL.—The ordinary type consists of sudden loss of consciousness. The patient suddenly stops what he is doing, the face becomes pale and fixed, the pupils dilate; but after a few seconds consciousness is regained and the patient resumes his work or conversation as if nothing had happened. Auræ are infrequent in petit mal. In some instances the attack consists of forced movements, such as the sudden running forward of procursive epilepsy. In other cases, during the attack the patient may perform some automatic action, such as undressing himself or tearing to pieces whatever may be within reach. There may be sudden outbursts of maniacal excitement during which crimes and assaults may be committed: these cases of "masked epilepsy" are of great medico-legal interest. Somnambulistic epilepsy consists in the performance of accustomed acts while in a somnambulistic state.

Attacks of petit mal terminate in some instances in facial twitchings or in hysteroid convulsive movements. The various manifestations of petit mal are exceedingly varied. In the majority of cases attacks of grand mal ultimately develop, and the two forms may alternate.

3. JACKSONIAN EPILEPSY, which is regularly due to irritative lesions of the motor centres, especially of the motor cortical zone, differs from true epilepsy in the fact that consciousness is retained.

The spasm occurs in limited groups of muscles, which are always the same in each patient. Preceding the attack there may be numbness or tingling of the affected part. In growing lesions the march of the spasm may be observed, and accurate localization becomes possible.

Diagnosis.—Petit mal may be mistaken for syncope, vertigo, or indigestion, but in these conditions consciousness is not lost. Jacksonian epilepsy is rarely mistaken for other conditions. Localized spasms may, however, occur in uræmia and in progressive paresis.

Grand mal is to be diagnosed from uræmia, simple convulsions in children, convulsions from organic brain disease, malingering, and hystero-epilepsy. Uræmia is diagnosed by the high arterial tension, the scanty and albuminous urine, and the presence of fever during the attack.

Simple convulsions in children are usually due to some recognized cause and are not apt to be repeated. Convulsions due to organic brain disease, such as tumors of the cerebellum and progressive paresis, are to be diagnosed by attention to the history and the other symptoms. Malingerers may closely simulate epilepsy, but the tongue is not bitten, foaming at the mouth does not occur, and strong pressure by the thumbs over the supraorbital notches will rapidly cut short the attack. The diagnosis from hystero-epilepsy is to be made by the following points, thus tabulated by Gowers:

	Epileptic.	Hysteroid.
Apparent cause	None.	Emotion.
Warning	Any, but especially unilateral or epigastric auræ.	Palpitation, malaise, choking, bilateral foot-aura.
Onset	Always sudden.	Often gradual.
Scream	At onset.	During course.
Convulsion	Rigidity followed by "jerking," rarely rigidity alone.	Rigidity or "struggling," throwing about of limbs or head, arching of back.
Biting	Tongue.	Lips, hands, or other people or things.
Micturition	Frequent.	Never.
Defecation	Occasional.	Never.
Talking	Never.	Frequent.
Duration	A few minutes.	More than ten minutes, often much longer.
Restraint necessary	To prevent accident.	To control violence.
Termination	Spontaneous.	Spontaneous or induced (water, etc.).

The **prognosis** for cure, except in Jacksonian epilepsy, is bad, but the disease may be materially relieved by treatment. The prognosis is better in cases coming on in adults, due to syphilis, and in children in cases where the convulsions have followed teething or acute fevers. Epilepsy does not tend materially to shorten life.

The question of the intellectual future of the patient is always a serious one. Mental degradation occurs in a considerable proportion of cases, but epilepsy is not necessarily incompatible with an active and useful life.

Treatment.—In cases of reflex or of Jacksonian epilepsy the cause of the irritation should be removed. In some cases the results are brilliant, but in many instances there is but partial improvement, the habit of nerve-discharge of the higher centres having been established. In cases of epilepsy in which the aura is slow, attempts should be made to check the spasm by the inhalation of amyl nitrite; or in case of ascending sensory aura of an extremity, the patient should be taught to encircle the part firmly with the hand or with a tight bandage. Unfortunately, the auræ are usually of too short duration to allow of any preventive treatment.

During the attack the patient should be placed in a horizontal position, the clothing loosened, and a gag firmly placed between the teeth, to prevent the tongue from being bitten. Inhalations of chloroform or of ether are permissible in protracted or severe paroxysms, or a hypodermic injection of morphine may be administered.

Dietetic and Hygienic Treatment.—The patient should do all things in moderation, never in excess. Marriage should be interdicted. Habits of firm but kindly discipline are important for growing epileptic children. The diet should be chiefly, but not altogether, vegetable; meat, however, may be allowed once a day. The patient should be restrained from going to bed until gastric digestion has been completed.

Medicinal Treatment.—Bromides are the most serviceable drugs in the treatment of epilepsy. Of the various bromides, the salt of sodium is the most preferable. The bromide treatment should be pushed until mild affects of bromism—acne, mental depression, foul breath, and muscular weakness—have been produced, and should then be reduced so that the patient is kept just within the physiological action of the drug, so that the palate-reflex is lost. As a rule, from ½ to 1½ drams daily are sufficient for an adult. The drug should be largely diluted in water or in milk, and the liability to acne is said to be diminished by the joint administration of arsenic. The bromide treatment should be continued for at least two or three years after the cessation of the attacks.

The combination of antipyrine with the bromides is frequently of service. Wood claims that a mixture of bromide of ammonium (gr. xx–xxx), antipyrine (gr. vij), and Fowler's solution (♏ ij–iij) affords the best combination known for the majority of cases, the indicated doses being administered twice daily. Sulphonal may also be given advantageously with the bromide treatment.

Among other forms of treatment employed for their alleged specific action are valerian, belladonna, oxide of zinc, and nitrate of silver and borax, but these drugs prove almost worthless when tried.

PARALYSIS AGITANS.

Etiology and Synonyms.—Paralysis agitans usually appears between the fiftieth and sixtieth years, and is rare before the age of forty. Men are more commonly attacked than women, in the proportion of 5 : 3. Hereditary influences can be traced in 15 per cent. of all cases. In about one-third of all cases an exciting cause can be ascertained—emotion, fright, exposure to cold, physical fatigue, or acute disease. In other cases the disease begins without known cause. It is not a disease of vice. *Synonyms:* Shaking palsy; Parkinson's disease.

Pathology.—No lesions are found to account for the condition, but it is supposed that the symptoms are due to premature senile changes in the cerebral cortex.

Symptoms.—The characteristic symptoms are tremor and muscular rigidity.

The *tremor* develops insidiously, and usually appears first in the hands or the fingers. At first the tremor may be controlled by the will, but later it becomes more continuous and cannot be controlled. The tremors are short, rapid (being about 5 to the second), and in the fingers may be rhythmical, so that the motion resembles that produced by rolling some small body between the thumb and the fingers. The handwriting shows the character of the fine tremors. It is peculiar for the tremor to continue when the hand or the limb is at rest and to cease during voluntary motions, so that the patient may safely carry a glass of water to the lips. In other cases the tremor cannot be thus checked, and in rare instances it may even be increased by voluntary motions. Emotions regularly increase the tremor. The tremor extends to various parts of the body without fixed order of progression, but the face is rarely involved. Voluntary motions are performed slowly and with but little power.

Muscular rigidity, which is characteristic of the advanced stages of the disease, gives rise to changes in the attitude, the gait, and the facial expression. The attitude is characteristic (Fig. 68); the body is inclined forward, and the extremities are in a general condition of flexion. The inclination of the body forward may throw the patient in front of the centre of gravity, so that he will have to walk faster and faster, or even to run, to avoid falling forward—the so-called "festinating gait." The face is fixed, expressionless, and immobile; the eyebrows are raised, giving a characteristic facies to which the name "Parkinson's mask" has been applied. The voice is a high-pitched monotone.

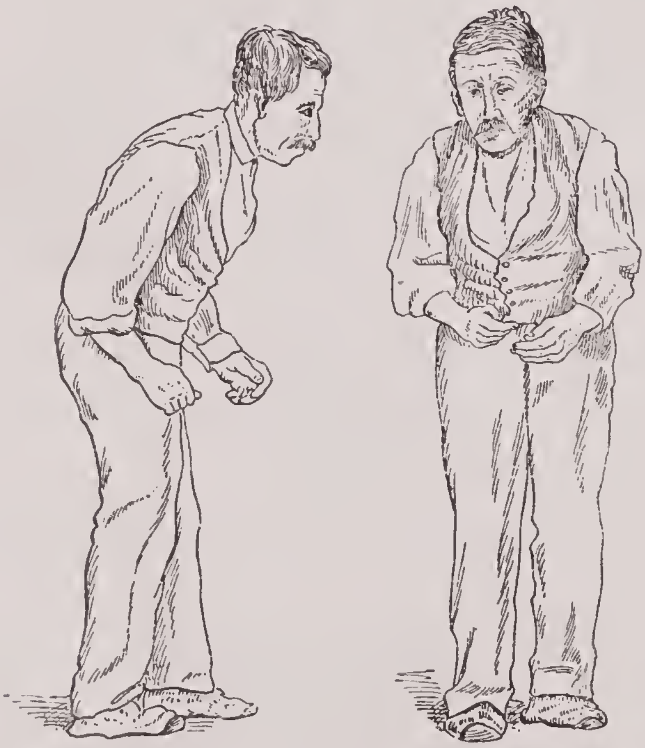

FIG. 68.—Paralysis agitans (St. Leger).

Sensory symptoms consist of soreness and a sense of fatigue in the affected muscles. Various vaso-motor symptoms may occur. The surface-temperature over the affected muscles may be increased. Mental derangement does not occur, although the patients may become emotional. The urine may contain an excess of phosphates, or there may be polyuria.

Diagnosis.—Disseminated sclerosis develops earlier in life, nystagmus is present, the speech is scanning, and there is no characteristic attitude. The diagnosis from post-hemiplegic tremor is readily made by the history of the case, the increased reflexes, and the hemiplegic distribution of the latter disease. Senile tremor is rare under the age of seventy, and is usually marked in the muscles of the neck, producing slight movements of the head. Toxic tremors from alcohol or from tobacco usually occur only on motion, and the tremor is more pronounced, showing considerable range.

Prognosis.—The condition is incurable, but it does not tend to shorten life.

Treatment.—A number of drugs have been recommended, but no form of treatment seems to have any permanent influence upon the disease. Mental and physical rest should be enjoined, and prolonged lukewarm baths may be advised. Hyoscine hydrobromate (gr. $\frac{1}{100}$ gradually increased) has been given with temporary benefit. Dana has used the bromide of uranium (gr. $\frac{1}{80}$) with apparently good results. Arsenic may also be used.

ACUTE DELIRIUM.

Etiology and Synonyms.—Acute delirium usually occurs during active adult life, and may be due to alcoholism, to profound grief, or to over-work; or the condition may appear as a sequel to sunstroke, fevers, or injuries to the head. *Synonyms:* Bell's mania; Acute periencephalitis.

Pathology.—The nature of the disease is unknown. By some authors the lesion consists of hyperæmia and œdema of the brain and its membranes, with a choking of the lymph-channels of the pia and of the cortex by leucocytes.

According to others, the symptoms are due to nerve-poisoning by unknown toxic products.

Symptoms.—The onset may be preceded by the prodromal symptoms of irritability, restlessness, and insomnia. The developed disease presents two stages, one of maniacal delirium, the other of apathy, collapse, and coma. The delirium comes on rapidly and reaches a grade of wild frenzy with hallucinations and delusions. There is absolute insomnia. The temperature ranges between 102° and 105° F., but falls to subnormal in the advanced stages. The stage of mania lasts for from a few hours to several days and is succeeded by a stage of quiet in which the patient lies semi-comatose, responding incoherently when aroused. In the advanced stage the pulse fails and the symptoms of collapse become evident. There may be irregular desquamation of the skin, ulceration, gangrene, pemphigus, or areas of complete anæsthesia.

The **diagnosis** should be made from the following conditions: (1) Masked pneumonia with maniacal delirium at the onset; (2) acute uræmia with mania followed by coma; (3) typhoid fever with marked cerebral symptoms at the onset; (4) delirium tremens; (5) acute meningitis.

Prognosis.—The duration of the disease is about a week, but it may be protracted for two or three weeks. The disease is almost uniformly fatal. When recovery occurs the mind is almost regularly affected.

Treatment.—During the maniacal stage the patient should be actively purged, and in robust cases free venesection should be resorted to. The patient is to be quieted by morphine, hyoscine, or chloral, and when tractable the cold bath or cold pack may be employed for its calmative effect. Good results are claimed for the hypodermic use of ergotine in large doses, 15 grains being given every eight hours.

CHOREA.

Etiology and Synonyms.—The disease is more common in females than in males, and three-fourths of all cases occur between the ages of five and fifteen. Chorea is rare before the fourth year. Negroes are usually exempt. The disease is most common in high-strung, nervous, excitable children, especially those who are ambitious at school. The exciting cause in 15 per cent. of all cases is fright, but mental worry, grief, or sudden trouble may also precipitate an attack. Ocular defects and reflex irritation do not seem to produce true chorea. An antecedent history of rheumatism is obtained in about one-fifth of the cases. In only 2 per cent. of the cases does chorea antedate the rheumatic complaint. Chorea may occur after certain infectious diseases—scarlet fever, whooping-cough, gonorrhœa, secondary syphilis, and septic infections—and may also complicate pregnancy, especially in primiparæ during the first five months. *Synonyms:* St. Vitus's dance; Sydenham's chorea.

Pathology.—There are no characteristic lesions in chorea. In some cases vascular changes are found in the nervous system—hyaline transformation, exudation of leucocytes, small punctate hemorrhages, and thrombosis of the smaller arteries—but these changes are inconstant. Endocarditis is the most frequent lesion found at autopsies, occurring in about 90 per cent. of the cases. The endocarditis may be of the malignant variety.

The *nature of the disease-process* in chorea is not yet accurately known; three theories for its causation are advanced: (1) That chorea is a functional brain disorder, leading to an instability of the nerve-cells controlling the motor apparatus, induced by hyperæmia or anæmia, by psychical influences, or by central or reflex irritation. (2)

That chorea is due to embolism of the smaller cerebral arteries. This view is favored by the experimental production of chorea in animals by injecting fine insoluble particles into the carotids. All cases of chorea, however, cannot be thus explained, as histological examination of the brain may be negative. (3) That chorea owes its origin to a microbic infection at present unknown. This view seems highly probable, although it does not explain why chorea is so frequently excited by fright or other psychical disturbance.

Symptoms.—The disease may begin abruptly or may be preceded by prodromata. The child becomes restless, is fidgety, irritable, and emotional.

Three grades of the developed disease are described by Osler:

1. *Mild chorea,* in which the muscular affection is slight, speech is not seriously affected, and the general health remains good. In the mild cases there are constant irregular, jerky motions involving usually a hand or a hand and the face. There may only be awkwardness or slight inco-ordination of voluntary motions.

2. The *severe form*, in which the choreic movements become general, and are so pronounced that the patient cannot get about, feed, or undress himself. The speech is usually affected, and there may be motor weakness on one side or in the limb most affected.

3. The *maniacal form*, or *chorea insaniens*, characterized by profound cerebral disturbances and by violent choreic motions. There is active delirium, and there may be hyperpyrexia, especially in the fatal cases.

The individual symptoms may be thus described:

1. *Motor symptoms* may not pass beyond awkwardness or slight inco-ordination, or they may develop into unwilled clonic spasms of various parts. The hands are usually

first involved, then the face and subsequently the legs. The movements may be confined to one side—hemichorea. The attack usually begins on the right side, but in rarer instances the movements may be general from the start. The muscles of the trunk and of the thighs are usually bilaterally affected.

In about one-fourth of the cases the *speech* becomes affected, from involvement of the lips and tongue. In mild cases there is merely embarrassment or hesitancy; in severer cases articulation becomes jumbled and incoherent, so that the child ceases from attempts to talk. The inspiratory muscles may be affected, so that the patient will emit sighing or odd barking sounds. Choreic movements generally cease during sleep. There is no evidence that the muscles of the gastro-intestinal tract, the bladder, the rectum, or the bronchi are ever affected, and irregular contractions of the papillary heart-muscles probably do not exist.

Muscular weakness is not uncommon, and rarely amounts to more than an enfeeblement of the grip or a dragging of the foot. In other cases there may be more evident paresis, either of the hemiplegic, paraplegic, or monoplegic type ("paralytic chorea").

2. *Sensory Disturbances.*—Pain in the muscles of the affected limbs is uncommon, but pain and tenderness along the nerve-trunks may be marked, especially in cases of hemichorea. Numbness and tingling or pricking sensations are occasionally encountered. Multiple neuritis may occur. Headache is frequent and may be persistent.

Mental symptoms are rarely absent, although rarely are they pronounced. There may be irritability of temper with emotional outbreaks, and a change in the moral character of the child. Memory and aptitude for study are impaired. Melancholia is not uncommon. In rare instances dementia develops. Aggravated cases of chorea, the chorea insaniens, are characterized by delusions, hallucinations, delirium, and even mania.

The *reflexes* in chorea may be normal; in one-half the cases the knee-jerk is diminished or lost. Cutaneous affections are common in chorea, but are generally due to the arsenic treatment or to allied rheumatic affections. The skin affections embrace erythematous and papular pigmentations, rashes, herpes zoster, erythema nodosum, and purpura rheumatica. Fever occurs in about one-eighth of all cases, and is usually slight. Any febrile condition other than a slight transient rise of temperature is indicative of some complication—rheumatic arthritis, pericarditis, or endocarditis. Fatal cases of chorea insaniens are usually accompanied by a high ante-mortem temperature.

Heart Symptoms.—Neurotic palpitation is not uncommon. Cardiac murmurs occur in from 25 to 30 per cent. of all cases, and may be either of hæmic or of organic origin. Hæmic murmurs are most commonly observed between the third and fourth weeks. Acute choreic endocarditis rarely gives rise to symptoms. The following statements are given by Osler:

"In thin, nervous children a systolic murmur of soft quality is extremely common at the base, particularly at the second left costal cartilage, and is probably of no moment.

"A systolic murmur of maximum intensity at the apex, and heard also along the left sternal margin, is not uncommon in anæmic, enfeebled states, and does not necessarily indicate either endocarditis or insufficiency.

"A murmur of maximum intensity at apex, with rough quality, and transmitted to axilla or angle of scapula, indicates an organic lesion of the mitral valve, and is usually associated with signs of enlargement of the heart.

"When in doubt it is much safer to trust to the evidence of eye and hand than to that of the ear. If the apex-beat is in the normal position, and the area of dulness is not increased vertically or to the right of the sternum, there is probably no serious valvular disease.

" The endocarditis of chorea is almost invariably of the simple or warty form, and in itself is not dangerous; but it is apt to lead to those sclerotic changes in the valve which produce incompetency. Of 110 choreic patients examined more than two years after the attack, 54 presented signs of organic heart disease.

" Pericarditis is an occasional complication of chorea, usually in cases with well-marked rheumatism."

The **diagnosis** is usually evident. Chorea should not be mistaken for spastic diplegia in children, for cerebral atrophy, or for Friedreich's ataxia. In the former case the onset in infancy, the steady and chronic course, increased reflexes and muscular rigidity, and the impaired intellect are distinguishing features. In Friedreich's ataxia the scanning speech, spinal curvature, nystagmus, deformities of the feet, and the slow, inco-ordinate movements render the diagnosis easy. Hysteria may closely resemble chorea, but the movements are rhythmic and not choreiform in character, and other hysterical symptoms are present.

Prognosis.—Recovery is the rule, although there exists in chorea a tendency to recurrences, especially in rheumatic cases. Recurrences are most frequent during the spring months. The total mortality is about 2 per cent. The mildest cases get well in two or three weeks. The ordinary duration is about two months, but cases may drag along for from three to six months. The ultimate prognosis, however, is that of the associated cardiac lesions.

Treatment.—Excessive brain-work and eye-strain at school, and the competing for prizes, should be prohibited in nervous children, especially in those who have had previous attacks of chorea. The general nutrition of the child should be maintained, and anæmic conditions should promptly be met by the administration of iron and arsenic. For the attack itself rest and seclusion constitute important elements in the treatment, and are insisted upon by Osler. The child should be put to bed and kept quiet until the movements have ceased. By this procedure the liability to heart complication is materially diminished. The child should be kept quiet, and should not be excited by toys or by seeing too many people. The diet should be nourishing and abundant. Arsenic is the best form of medicinal treatment, and is given as a matter of routine practice. Children bear the drug well. Fowler's solution is to be given in 3-minim doses well diluted, after meals; the dose should be increased by 2 minims every second or third day until from 12 to 15 minims are taken at each dose. Should toxic symptoms appear—vomiting, diarrhœa, itching of the eyelids, œdema, or skin affections—the drug should be stopped for three or four days and then be resumed at the same dose as that last taken. According to Osler, arsenic seems to exert no specific action upon the disease, but does good by improving the general condition.

Of other remedies, cimicifuga, chloral, sulphonal, physostigmine, antipyrine in 20- to 60-grain doses throughout the day, and quinine in large doses have been recommended. The zinc compounds, strychnine, and sodium bromide may also be tried in obstinate cases. Iron is required in nearly all cases to combat anæmic conditions.

For chorea insaniens hydrotherapy, in the form of the wet pack or the bath, should be tried, and the patient should be quieted by chloral, morphine, or, in the severest cases, by whiffs of chloroform. The cardiac affections are to be treated on general principles. Obstinate cases may be benefited by change of air and by enforced rest and seclusion.

Choreiform Affections.

Habit-spasm (Habit-chorea; Simple Tic).—This condition is common in childhood, and may persist during life. The patients are frequently over-grown children of a neurotic personal or family history. There may be twitching of the eyelids, facial grimaces, shrugging of the shoulders, or short inspiratory sniffs. In severer forms nearly all the muscles of the face are affected. A "generalized tic" occurs in children and in adults and may persist for years. The muscles of the extremities and of the trunk suddenly jerk, producing the effects of a general electric contraction ("electric chorea").

Tic Convulsif (Gilles de la Tourette's Disease).—In this form which usually occurs in nervous children with a neurotic family history, in addition to motor spasms there occur explosive utterances of sounds or of words. A sound may be repeated over and over again (*echolalia*), or obscene and profane words may be used (*coprolalia*). In some cases fixed ideas are present; of these, *arithmomania* is the most common, in which condition the patient feels obliged to count a certain number of figures before almost every action.

Huntingdon's Chorea (Chronic Chorea).—This rare disease is characterized by its hereditary nature, a tendency to insanity and suicide, and its late onset between the thirtieth and fortieth years. When one or both parents have been subject to the disease, one or more of the offspring invariably become affected; but the hereditary character of the disease is peculiar in that it never skips one generation to manifest itself in another. The pathology of the disease is obscure.

The symptoms are first manifested in the hands by irregular movements; later the movements are disorderly and inco-ordinate, and have not the jerky character of the true choreic contractions. Slow involuntary facial grimaces occur; the gait becomes swaying and irregular, and has been aptly compared to that of a drunken man. The arms and hands are usually in more or less constant irregular motion. The speech is affected in the majority of cases, becoming slow, hesitating, and indistinct. Mental impairment becomes progressively marked, and ultimately terminates in dementia.

TETANY; TETANILLA.

Etiology.—This condition, which is rare in the United States, most commonly occurs before the twentieth year. In the great majority of cases an exciting cause can be discovered—exposure to cold, acute diseases, especially typhoid fever, fatigue, lactation ("nurse's contracture"), or pregnancy. In young children the indications of rickets are seldom absent. Tetany occurs in about one-sixth of the cases of removal of the thyroid gland, and may be fatal. Epidemics of tetany are described as occurring on the Continent of Europe, and appear to be due to some unknown infection. A rare but fatal form complicates dilatation of the stomach.

The **pathology** of the disease is unknown. It is probable that the disease depends upon the action of some toxic agent upon the motor centres.

Symptoms.—The spasms are bilateral, and begin in the hands and feet. The fingers are flexed at the metacarpophalangeal joints, extended at the others; the thumb is flexed and adducted; the palm is hollowed. The wrist is flexed, and the arm may be folded over the chest. The feet are extended and inverted; the toes are flexed. In severe cases the muscles of the trunk and of the face may be involved, and there may be trismus. Dyspnœa and cyanosis may result from spasm of the respiratory muscles. The spasms are usually paroxysmal, lasting from several minutes to several hours or even days, but in some severe forms the symptoms are continuous for several weeks. In the acute forms there may be a moderate fever, a feeling of "pins-and-needles" in the hands, and a cramp-like pain in the affected muscles. During the height of the paroxysm, and persisting for several weeks afterward, there is a greatly increased excitability of the affected nerves to the galvanic and the faradic current, tetanic contractions following the application of a current which in health would produce no appreciable reaction. The slightest tap on the affected muscle causes also a conspicuous contraction. The characteristic spasm may also be induced by pressure on the artery, sometimes by pressure on the nerves of the limb ("Trousseau's phenomenon").

Diagnosis.—From tetanus the disease is distinguished by the fact that the earliest symptom in tetanus—trismus—is the latest in tetany. Hysterical contractures are almost invariably unilateral, while tetany never is. Cases of carpopedal spasms in rickety children should not be regarded as cases of true tetany.

The **prognosis** is favorable except in those cases following thyroidectomy or dilatation of the stomach. Future attacks are liable to occur, however, if the exciting cause be repeated.

Treatment.—The cause of the disease should be traced and removed. Lactation should be stopped, and in all cases a tonic form of treatment, with baths and cold sponging, should be advised. Sodium bromide relieves the spasm most effectively, but cannabis indica or chloral may also be used. Ice to the spine has been recommended. Electrical treatment is disappointing. Faradism is contraindicated. Massage under chloroform-narcosis has been followed by good results in obstinate cases.

MIGRAINE.

Etiology and Synonyms.—This affection, which is often inherited, is more common in women and in members of neurotic families. In many of the cases there is a history of rheumatic or gouty taint. The existing cause may be mental or bodily fatigue, emotions, indigestion, or the eating of some particular article of food. Among reflex causes should be mentioned uterine disease, eye-strain, abnormal conditions of the nose or of the naso-pharynx, and carious teeth. A reflex source of irritation should always be suspected in the migraine of young patients. The attacks often appear with striking periodicity, and usually cease after the climacteric, or in men after the fiftieth year. *Synonyms:* Hemicrania; Sick headache.

Pathology.—The nature of the disease is unknown. Liveing's theory is that it is a nerve-discharge from sensory centres—the sensory equivalent of epilepsy; according to others the disease is a vaso-motor neurosis.

Symptoms.—Premonitory symptoms are present in many cases. There may be malaise, lassitude, and a sense of chilliness. Visual prodromes are not uncommon—hemianopia, spots of dimness of vision or scotomata, apparitions, balls or flashes of light, and zigzag lines. Sensory prodromes consist in numbness or tingling of a hand or an arm, or of peculiar sensations in any part of the body. There may be a condition of intense emotional activity. Motor prodromes consist in temporary weakness of certain groups of muscles or of aphasia.

The prodromal symptoms are not always present. In some cases they may comprise the entire attack, not being followed by headache.

The characteristic symptom of migraine is the violent paroxysmal headache.

Beginning over one side, usually most intense over the frontal region or over the eye, the pain grows more and more unendurable. In rarer instances the headache is bilateral.

The pain is usually described as of a sharp, boring character, and is regularly increased by the slightest sound or light. Prostration, though temporary, is extreme. During the early part of the attack the face may be pale and pinched, and there may be a marked difference between its two sides. Subsequently the face becomes flushed from vaso-motor dilatation. During the attack there is usually mental confusion, or even temporary loss of memory. The pulse may be slow and the temporal artery contracted and in a condition of arterio-sclerosis. When the headache reaches its climax nausea and vomiting commonly appear; the vomiting generally affords relief, so that the patient may fall at once into a sound sleep and awake refreshed.

The **duration** of an attack varies from several hours to several days.

Migraine is not accompanied by fever except in children, in whom a temperature of 102° to 103° F. may be developed.

Prognosis.—Much can be done to render the attacks less frequent and severe, but the disease cannot be radically cured by medication. Spontaneous cure usually occurs between the fortieth and fiftieth years.

Treatment.—Each patient is usually aware of the causes that precipitate an attack, and if these causes be avoided the paroxysms are rendered much more infrequent. Peripheral irritations, such as eye-strain, nasal hypertrophies, or adenoid growths in the naso-pharynx, require appropriate treatment. Attacks of migraine become infrequent during good health; the patient therefore should be built up, and gouty and rheumatic tendencies should be corrected. During the paroxysm the patient should be put to bed and kept absolutely quiet. A small cup of strong hot coffee frequently affords relief. Much benefit is derived from the use of antipyrine or phenacetine, especially when given in small repeated doses. If there be conspicuous pallor, nitroglycerin in does of gr. $\frac{1}{100}$ may be given every two hours. Cannabis indica (gr. $\frac{1}{3}$, Herring's English Extract), sodium bromide (gr. xxx), and chloral hydrate (gr. x–xv) are all of service. A prolonged course of cannabis indica is frequently beneficial in reducing the number of attacks. Of other remedies, caffeine, guarana, ergot, and sodium salicylate have been recommended.

OCCUPATION-NEUROSES.

Etiology.—Certain localized motor affections occur in those whose occupation requires the constant repetition of complicated muscular movements. The most common form is "writer's cramp," but piano- and violin-players, telegraphers, and cigarette-rollers may be similarly affected. Men are more frequently attacked than women. Predisposition is afforded by any of the causes leading to neurasthenia.

Pathology.—The condition is one of local neurasthenia, the affected nerve-centres being in a condition of "irritable weakness."

Symptoms.—The principal symptoms are pain and spasm. A paralytic form has also been described. The symptoms of "writer's cramp" may be taken as a type. There is a feeling of fatigue in the affected muscles, amounting to actual pain when writing is attempted; tremor and irregular spasmodic contractions occur, rendering the writing illegible; later the pain may be more continuous, may spread over the arm, and may be accompanied by tenderness along the course of the nerve-trunks.

The **prognosis** must be made guardedly, as the condition tends to become chronic, and even when relieved by treatment the affection is liable to recur.

Treatment.—It is important that in writing the motions should be made from the arm or the forearm, and not from the wrist or the little finger, as the fixed point. When the symptoms are first noticed rest is imperative. The various devices of complicated pen-holders only serve to stave off the disability and allow the patient to do his work while the malady is really getting worse. Massage and systematic gymnastics are of service. Electricity does not seem to do good. No form of treatment, however, is of benefit that is not combined with rest. The general nutrition of the patient should be improved in every way. Nervine tonics, as phosphorus and strychnine, are to be recommended.

NEURASTHENIA.

Etiology.—Nervous weakness and irritability occur when the expenditure of energy exceeds its supply. The following classification of causes is given by Starr:

A. *Excessive expenditure of nerve-energy* (primary neurasthenia): (1) Bad hereditary influences, weak nervous system; (2) feebleness in childhood, with poor nervous system; (3) wrong methods of training; (4) the struggle for existence; (5) anxiety, mental depression, worry, fear; (6) mental or physical over-work; (7) sexual excesses.

B. *Deficient supply of nervous energy* (secondary neurasthenia): (1) Weakening diseases of all kinds, of an organic nature; (2) indigestion and dyspepsia, with auto-infection by toxic products; (3) gout, rheumatism, uric-acid diathesis; (4) infectious diseases—typhoid, grippe, malaria; (5) alcoholism and the abuse of drugs.

Pathology.—There are no anatomical lesions. The condition consists of "irritable weakness" of the nervous centres.

The **symptoms** of neurasthenia are so varied that only a brief description can be given of the important manifestations of the disease. The various symptoms are grouped in patients in an infinite variety of combinations.

Cerebral and Mental Symptoms.—There is an inability to perform the ordinary mental work. The patient is moody, apprehensive, irritable, and depressed, and complains of a sense of fulness in the head, of throbbing, or even of actual headache. Suboccipital headache is common, and is usually associated with insomnia. The eyes are easily tired after reading for a few minutes.

Spinal Symptoms.—There are weariness on exertion, pain in the back, and tender spots along the spine. Pain over the cervical vertebræ and the sacrum is not uncommon. Sexual neurasthenia is characterized by nocturnal emissions, impaired power, and a distressing dread of impotence.

Vaso-motor symptoms consist of hot and cold flashes, localized sweatings, transient blueness, or œdema. Palpitation, irregular heart-action, and painful feelings in the heart are commonly present, and distress the patient with the belief that he has serious organic disease of the heart. Tachycardia may occur.

Gastro-intestinal Symptoms.—There may be nervous dyspepsia, or hyperacidity, or hypersecretion. Constipation is the rule. There may be membranous colitis, especially in women. Tympanites is a common symptom. Gastro-intestinal neurasthenia is often associated with dilatation of the stomach and floating kidney, the combined conditions being termed "enteroptosis."

Urinary Symptoms.—In many cases oxalate of lime or uric acid is present in the urine—the so-called "lithæmic neurasthenia." There may be polyuria.

The **diagnosis** is made by the grouping of a variety of neurasthenic symptoms without actual organic disease. In many cases the diagnosis is rather a question of medical intuition.

The **prognosis** is good if the exciting cause can be removed and if the patient is in such a financial condition as to be able to carry out the necessary treatment. Many patients, handicapped from birth with a weak nervous system, do well only when no demands are made upon their strength ; as soon as any emergency arises they go under.

Treatment.—The most important indication is to discover the cause ; this often requires an intimate and personal knowledge of each individual patient. Over-worked business-men are best treated by rest with a change of scene, as by a trip abroad. Pleasant physical exercise to the point only of moderate fatigue is often beneficial. The general nutrition should be improved in every way by proper food, sufficient sleep, and a proper amount of recreation, and by massage, hydrotheraphy, and tonics. The drug treatment of these patients should be limited, however, as much harm is done by over-dosing. Stimulants and narcotics are to be withheld, as habits are easily formed. In severe cases the Weir-Mitchell rest-cure may be recommended.

HYSTERIA.

Etiology.—This most perfect type of a functional malady is the product of advanced civilization. Unknown among barbarous nations, it reaches its highest development among the French people. Hysteria is twenty times more common in women than in men, and usually appears between the ages of fifteen and twenty, although manifestations may continue until late in life. In many cases there is a family history of nervous troubles. With or without such an inherited neurotic predisposition, hysteria may be induced by injudicious training, an unstable moral organization, or a lack of self-control. Among more direct influences are emotions, unhappy love-affairs, domestic worries, sexual excess, masturbation, and physical enfeeblement by injury or acute disease. In some cases ovarian or uterine disease seems capable of originating hysteria, but these cases are much less common than are ordinarily supposed. Hysterical symptoms may spread to other patients by sympathetic imitation, or "moral contagion" as it may be termed.

Pathology.—There is no organic lesion in hysteria; the condition is entirely functional.

Symptoms.—The clinical picture of hysteria is so varied and complex that only a brief description of individual symptoms can here be given, and no attempt will be made to evolve a general picture of the disease.

1. *Motor Symptoms.*—(*a*) *Convulsive Seizures.*—A mild and a severe form of hysteria are recognized. The mild form usually appears after emotions. The patient becomes "hysterical," laughs and cries alternately, and complains of a constricted feeling in the throat, as if a ball were rising into it ("globus hystericus"). There may even be painful sensations, referred to some internal organ, resembling sensory auræ. The patient then falls into convulsions, but not as suddenly as in epilepsy. The convulsive movements are clonic and irregular. The attack subsides gradually, usually with the passage of flatus or of a large quantity of limpid urine, and the patient, as a rule, has no recollection of what has happened.

The severe form, or hystero-epilepsy, is not as common in America as in France. The convulsions, which are usually preceded by minor hysterical manifestations, simulate true epileptic attacks; but they last longer, the tongue is not bitten, and the movements are not shock-like. The convulsive seizure is usually followed by emotional displays, by cataleptic poses, by opisthotonos or other distortions, and by attitudes and grimaces expressive of the deepest emotions. As the patient emerges from the condition delirium and hallucinations are not uncommon, or the patient may sink into a prolonged trance. The attacks may frequently be repeated, as in the *status epilepticus*, but, unlike that condition, do not seem to exert a deleterious effect upon the general health.

(*b*) *Paralysis* is common and may involve any part of the motor apparatus. The onset may follow a convulsive seizure or may be induced by emotion. The paralysis may be hemiplegic, paraplegic, or monoplegic. Hemiplegia is more common upon the left side, and is usually associated with hemianæsthesia. The face is not affected. Hysterical paraplegia is the most common form of paralysis, and the affected muscles may be flaccid or spastic. There may be only loss of power for certain combined movements (as walking), while the patient still retains the power to move the legs in bed. The reflexes may be increased, there is irregular voluntary resistance to passive motion, and a spurious ankle-clonus may be present. The muscles do not waste, the electrical reactions are normal, and bed-sores do not occur. The feet are usually extended and inverted. In some cases ataxia occurs, with paresis. Other hysterical manifestations are usually combined with paresis, forming a more or less characteristic symptom-complex. A characteristic form of hysterical paralysis is aphônia, in which the voice is lost or reduced to a whisper. Examination shows lack of approximation of the vocal cords during phonation, although the glottis can readily be closed by coughing. In many cases hysterical aphonia complicates acute catarrhal laryngitis.

(*c*) *Contractures and Spasms.*—Contractures may be hemiplegic, paraplegic, or monoplegic in type. The smaller joints are usually flexed, the larger joints extended. Ankle-clonus, exaggerated reflexes, and a spastic gait are commonly present, so that the case may resemble lateral sclerosis so closely that a differential diagnosis may be impossible. The contractures disappear, however, during sleep and during chloroform-narcosis, and, although usually protracted, tend eventually to disappear spontaneously. Hysterical trismus is not uncommon. Contractures of certain of the abdominal muscles, combined with relaxation of the recti and the inflation of the intestines with gas, produce "phantom tumors," which in some instances are associated with the symptoms of spurious pregnancy or even of labor. Phantom tumors readily disappear under full anæsthesia.

Clonic spasms are not uncommon, and are generally rhythmic in character ("rhythmic chorea," "hysterical chorea"). Volitional tremor may exist, resembling that of insular sclerosis.

2. *Sensory Symptoms.*—(*a*) *Anæsthesia* is exceedingly common, occurring usually in irregular areas which may also be insensitive to touch and devoid of the muscular sense. Hysterical hemianæsthesia is so characteristic as to possess positive diagnostic value.

(*b*) *Hyperæsthesia* may give rise to spontaneous pain, or merely to tenderness upon palpation. Hyperæsthetic areas are rare on the extremities, but are common on the head, especially over the sagittal suture. This pain over the vertex, of an agonizing character, is likened to the pain which would be experienced if a nail were being driven into the head, hence the term *clavus hystericus* which has been applied to it. Other hyperæsthetic areas are commonly found over the sternum, under the mammæ, along the vertebral column, and over the ovaries. If pressure applied to these hyperæsthetic zones induces hysterical manifestations, the zones are frequently spoken of as "hysterogenic points." Spinal hyperæsthesia may affect the whole column or only a single segment, and is often so extreme that the slightest touch gives rise to exquisite pain. Abdominal hyperæsthesia may simulate gastric ulcer, appendicitis, or peritonitis. In the latter case the resemblance is almost perfect, even to the presence of fever and the peritoneal facies. Hyperæsthesia of the breast may be accompanied by a diffused swelling of the gland, but the hysterical breast may be recognized by the exquisite superficial tenderness, by constant variations in the swelling, and by the recurrence of the symptoms at the menstrual period or after exceptional excitement or fatigue.

3. *Special Senses.*—There may be limitation of the visual field, especially for colors, or there may be hemianopia. The color-sense may be partially or completely lost (hysterical achromatopsia). Loss of the senses of smell and taste are not uncommon. Hysterical deafness may occur alone or may alternate with attacks of hysterical blindness. Hyperæsthesia of the eye or the ear may also occur, the former being the more common.

4. *Digestive Symptoms.*—The globus hystericus, or the feeling as though a ball were rising in the throat, may be associated with pharyngeal spasm. The spasm may spread to the œsophagus, so that swallowing becomes difficult or impossible (see Spasmodic Stenosis of the Œsophagus). Hysterical vomiting, in which the food is regurgitated soon after eating, and without attendant nausea, may persist for months without marked failure in nutrition. An antagonism to food (*hysterical anorexia*, or *anorexia nervosa*) may occur in young girls, and may be so marked that no food is taken for days; the patient finally becomes emaciated to an extreme degree, and death from asthenia may result in rare instances. Deception, however, is practised in the vast majority of these "fasting girls," food being taken unobserved. Depraved appe-

tite, dyspepsia, and gastric pains are not uncommon. Peristaltic unrest (see page 479) may also occur. Reversed peristalsis has occurred, the patient vomiting rectal enemata that have been previously given. Flatulency is common and distressing. Nervous diarrhœa may occur, diarrhœal movements usually being induced by eating. Constipation is more frequent, and may be so obstinate in character that the bowels do not move for weeks, despite energetic medication. Hæmatemesis due to vaso-motor disturbance may undoubtedly occur, but in every case deception should be suspected.

5. *Respiratory Symptoms.*—Rapid breathing (50 to 120 to the minute), without increased frequency of the pulse or other symptoms of dyspnœa, is characteristic of hysteria. Actual dyspnœa may attend laryngeal spasm, and suffocation may seem imminent. Hysterical aphonia has already been alluded to. Extraordinary cries and sounds, resembling those produced by animals, may be emitted. A dry, paroxysmal barking cough is a frequent symptom in chlorotic girls, and may spread in schools by unconscious imitation. Spurious hæmoptysis may occur, the sputum being usually pale-red in color rather than the crimson color seen in true hemorrhage. In these cases the blood comes from the mouth or the pharynx. In other cases deception is practised.

6. *Circulatory Symptoms.*—Cardiac irritability, palpitation, and pain over the heart are common symptoms. The cardiac pain may give rise to the symptoms of pseudo-angina pectoris. Hot and cold flashes, pallor, and flushings with a sensation of heat result from vaso-motor disturbance. Localized flushings, areas of circumscribed œdema, and similar phenomena may occur. Stigmata, or hemorrhages in the skin, may occur, although in the vast majority of cases they are of fraudulent origin.

7. *Urinary Symptoms.*—After hysterical manifestations the urine is abundant, limpid, and of a low specific gravity.

Retention of urine is common; incontinence is unknown. The urine may be partially or completely suppressed, and this hysterical anuria may last for days, during which time the sweat, vomit, and other secretions become loaded with urea. It is a characteristic of hysterical anuria that uræmic symptoms do not appear. Irritation of the bladder, shown by a constant desire to pass urine, is a frequent and troublesome symptom.

8. *Joint-symptoms* (Brodie's Joint).—The larger joints, especially those of the knee and the hip, are usually affected, and the symptoms may follow slight injuries. The joint is swollen, resists passive motion, and is flexed, although changes in position occur from day to day. The skin over the joint is hyperæsthetic and usually cool, but heat may be observed, especially at night, accompanied with pain. Motion and handling are painful. In protracted cases the muscles about the joint may waste. In rare instances organic changes in the joint may succeed the functional disturbance.

9. *Temperature.*—In hysteria the temperature is usually normal, although in severe cases a slight rise may be observed. Rare cases of "hysterical fever" are reported, in which a periodic elevation of temperature to 102° or 103° F. has been recorded. In very exceptional cases a rise to 108° or 110° F. has been noted, but these high temperatures are in all probability fraudulent. Cases of hysterical fever with spurious local manifestations are most deceptive. The occurrence of fever with hysterical symptoms resembling peritonitis has been described. Fever with pain in the head, photophobia, contracted pupils, vomiting, and retraction of the neck may closely resemble meningitis.

10. *Mental Symptoms.*—There is usually an exaggeration of ordinary emotional excitement. The moral character becomes changed; the patient becomes low-spirited and

listless or inquisitive and fussy. Often the patient's attention is morbidly concentrated upon herself. Whims of the most varied kind are invented from time to time; the patients show a marked tendency to deceive others, evince a wish to become objects of notoriety, and not the slightest dependence is to be placed upon their statements. Other patients become sullen and refuse to answer questions. Attacks of insanity and persistent hallucinations and delirium may occur. Trance or catalepsy may develop.

Prognosis.—The danger to life is so extremely small that it may be disregarded. The duration varies according to the severity of the symptoms, the moral force of the patient, the duration of the operative cause, and the home surroundings and environment.

Treatment.—It is essential that the physician should gain the patient's confidence from the start, and that he should enter into every detail of her daily life, so as to discover what elements are at work perverting her nervous forces. Hysteria arising from an unhappy home-life, from disappointments in love, and from kindred causes is best treated by travel and total change of environment. In many cases routine employment is to be recommended. The physician should not forget that the patient's complaints are not entirely imaginary, but that the suffering often is real. On the other hand, too much sympathy should not be given by either physician or friends. Many patients improve at once when they are taken from fussy and solicitous relatives and placed among strangers, where self-control must of necessity be exerted.

The general health must be improved in every possible way. The diet should be wholesome and abundant; exercise should be graded; and sufficient hours for rest and sleep must be insisted upon. Laxatives and other medication are indicated should dyspeptic symptoms be present.

Bromide of sodium, phenacetine, valerian, asafœtida, and similar antispasmodic drugs are of service during the acute manifestations of the disorder, but the continuous use of these remedies as a routine treatment is much to be deplored. Morphine should never be used.

In severe and intractable cases the Weir-Mitchell rest-cure—a combination of seclusion, rest, and forced-feeding—may be recommended, especially for under-nourished patients with marked hysterical manifestations, and for those who are constantly passing over the limits of their nervous strength. A bright, intelligent nurse and a complete isolation from family and friends are essential features of this treatment. Rest in bed for three or four weeks, with daily exercise of the muscles by faradism or massage, usually allows the strained nervous system to return to its normal condition and improves body-nutrition. The diet during the first ten days is to be milk alone; later, solid food is to be added gradually.

For hysterical vomiting and *anorexia nervosa* forced feeding by the stomach-tube is to be employed. During a convulsive attack a cold douche may be applied to the head, with the assurance to the family, in the hearing of the patient, that there is no danger, but that the cold applications will be continued until relief is experienced. In many instances, however, it is better to leave the patient alone and unnoticed until the attack subsides spontaneously. The treatment of hysterical paralysis by hypnotic suggestion has occasionally been followed by brilliant results.

SUN-STROKE.

Two distinct conditions due to exposure to intense heat are encountered—heat-exhaustion and thermic fever.

Heat-exhaustion may be induced by exposure to the hot sun or to any great artificial heat, as in engine-rooms.

There is prostration bordering upon collapse; the skin is cool and clammy; the face is pale; the pulse is rapid and feeble; the temperature is regularly subnormal. The onset of symptoms may be gradual, or may be so abrupt that the patient will fall in syncope. In severe cases the consciousness may be lost and muttering delirium may occur. It is thought that the condition is dependent upon a vaso-motor paresis as the result of which there is a determination of blood from the surface of the body and the brain to the large abdominal blood-vessels.

Treatment.—External heat should be applied, preferably by the hot bath. Stimulants should be freely administered.

THERMIC FEVER (Sun-stroke; Heat-stroke; Insolation; Coup de Soleil).—**Etiology.**—This condition is most frequent when great heat is combined with a high percentage of humidity. Excessive bodily fatigue and intemperance are predisposing factors.

Pathology.—Rigor mortis occurs early, and putrefactive changes appear rapidly. The blood is dark and imperfectly coagulated. There are parenchymatous changes in the liver and kidneys. The lungs are intensely congested.

The **symptoms** may begin abruptly, the patient falling unconscious to the ground; or they may be preceded by pain in the head, nausea and vomiting, vertigo, dimness of vision, or colored vision. When first brought under observation the patient is unconscious, with deep stertorous breathing and a rapid bounding pulse. The carotid arteries pulsate visibly. The skin is hot, dry, and reddish. Urine and feces are usually discharged involuntarily. The eyes are suffused, the pupils are variable, sometimes contracted, sometimes dilated. The temperature varies between 106° and 112° F., the average maximum height being about 108° F. Convulsions, muscular rigidity, and temporary delirium of an active form are of common occurrence. If the patient is to do badly, the pulse becomes rapid and irregular, the breathing becomes irregular or "Cheyne-Stokes," and death occurs, usually within from twenty to thirty-six hours. In very severe cases coma and heart failure may cause death within a few hours; in rarer cases instantaneous death may occur.

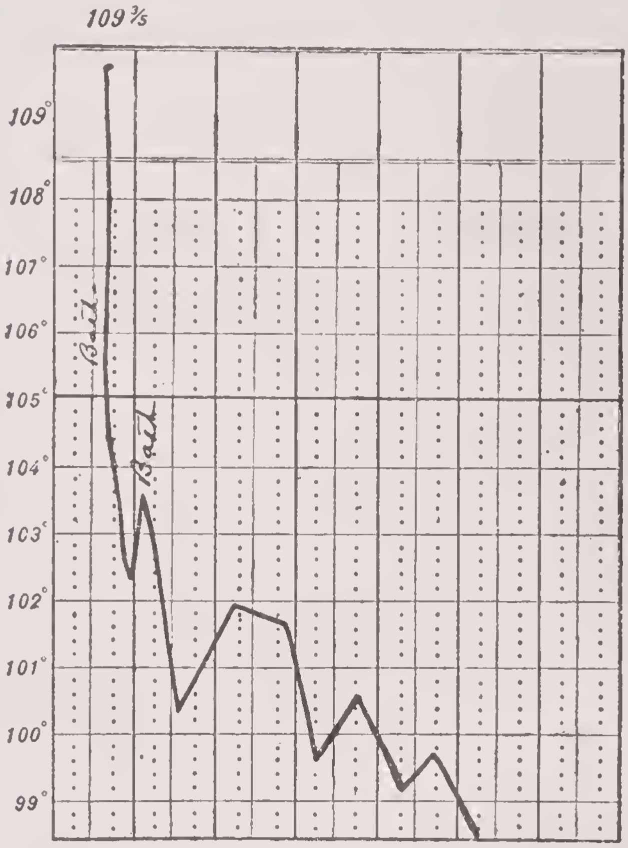

Fig. 69.—Temperature chart of sun-stroke.

A mild form of heat-fever, which occurs in tropical countries and gives the symptoms of a continued fever resembling typhoid, has been described under the terms "ardent continued fever" and "thermic continued fever."

Sequelæ consist of (1) inability to bear exposure to heat without headache and distress; (2) pain in the head, that may be constant for months; (3) mental weakness with nervous irritability. In rarer cases insanity or chronic meningitis may result.

The **prognosis** is grave. About one-third of the cases end fatally.

Treatment.—Immediate reduction of the body-temperature is most urgently indicated. Cold affusions and applications of ice to the surface must be resorted to without delay. When practical, the cold bath (50° to 60° F.) should be given; ice-water enemata may be serviceable. When a bath cannot be secured, the value of an ice-pack cannot be over-estimated. The cold applications are to be continued until the temperature is well under control. Internal antipyretics are not as serviceable as cold hydrotherapy. In very severe cases free venesection may be resorted to. In convulsions and delirium, morphine is to be given hypodermically. Stimulation is required in the majority of cases.

DELIRIUM TREMENS.

Etiology and Synonym.—Delirium tremens may be induced in hard drinkers by excesses, by sudden withdrawal of liquor, by fright, shock, or acute inflammation, especially pneumonia. *Synonym:* Mania à potu.

Symptoms.—The patient becomes tremulous, nervous, and is unable to sleep. Mental depression is extreme. After from one to three days the patient passes into the characteristic delirium, with visual and auditory hallucinations of a frightful character. The temperature ranges between 102° and 103° F.; the pulse is rapid and soft; tremulousness is often so extreme as to render speech incoherent; insomnia continues, so that the patient will get no sleep for three or four days. The condition either subsides in three or four days, especially after an induced sleep, or the insomnia persists, the pulse becomes more rapid and feeble, the delirium becomes of a low, muttering type, and death takes place from asthenia in a typhoid condition. Pulmonary congestion and œdema may complicate fatal cases.

Diagnosis.—It is important in every case to exclude local inflammations, especially of the lung, that may have been the exciting cause for the outbreak of the delirium. A careful examination of the lungs should be made every day, as delirium tremens may be simulated by the delirium of pneumonia at the apex of the lung.

The **prognosis** is generally good, only about 10 per cent. of the cases being fatal. Recurrences are to be expected unless the patient abandons the alcohol habit. Delirium tremens complicating pneumonia usually runs a fatal course.

Treatment.—The great object of treatment is to procure sleep and to support the patient's strength. The moderate use of opiates is recommended, but large doses are injurious. Alcohol should not be entirely withdrawn until after convalescence is established. Hydrotherapy is of service in many cases in controlling the delirium. Hyoscine in gr. $\frac{1}{100}$ doses hypodermically is of service in producing sleep. The combination of bromide of sodium, chloral, and tinctures of digitalis and capsicum is used in many hospitals, as in the following:

℞. Chloral hydrate,		gr. x;
Sodium bromide,		ʒ ss;
Tinct. digitalis,		
Tinct. capsici,	*āā.*	♏ v;
Spir. ammon. aromat.,		ʒj;
Aquæ,	q. s. ad	ʒij.—M.

Sig. Dose every two to three hours.

The patient should be constantly guarded by attendants night and day, to prevent his escape or injury. If necessary, he should be tied down by a draw-sheet, or in severe cases the strait-jacket may be applied.

7. VASO-MOTOR AND TROPHIC DISORDERS.

RAYNAUD'S DISEASE.

This vaso-motor neurosis presents three grades of intensity—local syncope, local asphyxia, and local or symmetrical gangrene. The majority of cases occur in young women.

Local syncope, the most common form, leads to coldness and pallor of the extremities ("dead fingers," "dead toes"), and may be induced by cold or by emotions. The affected parts are stiff, but are rarely painful. The condition lasts for a variable time and may be succeeded by local asphyxia.

Local Asphyxia.—There are lividity and cyanosis of the affected parts (the fingers, toes, ears, nose), with numbness, swelling, and some pain. The capillary circulation is exceedingly sluggish. The condition may follow that of local syncope. As the attack wears off, the affected parts become bright red from over-active circulation. Severe attacks may be followed by local gangrene. Recurrences are common, especially during the winter months.

Local or Symmetrical Gangrene.—Ecchymotic spots and vesicles appear, and become the starting-point for a dry gangrene which is usually remarkably symmetrical. The ears, fingers, and toes are the special seats of selection. The affection may be complicated by purpura hæmorrhagica, intermittent hæmoglobinuria, peripheral neuritis, and a variety of mental and cerebral symptoms.

Diagnosis.—The disease must not be confounded with the dry gangrene due to obliterating endarteritis.

The **prognosis** is good except in the extensive forms of gangrene or in the gangrenous cases complicated by purpura hæmorrhagica.

Treatment.—Exposure to cold should be avoided, and, if possible, the patient should spend the winters in a warm climate. The continuous electrical current may be applied to the spine and the extremities. Internal medication seems to exert no beneficial effect upon the disease.

ACUTE CIRCUMSCRIBED ŒDEMA.

Etiology and Synonyms.—This disorder, which is often of a distinctly hereditary character, may be induced reflexly by gastric disorders. Attacks occur in some instances with remarkable periodicity. Nothing further is known of its causation. *Synonyms:* Angio-neurotic œdema; Giant urticaria.

Pathology.—There is a localized vaso-motor dilatation allowing of the transudation of serum.

Symptoms.—A circumscribed œdematous swelling occurs in some part of the body, more commonly on the face or the arms. The skin is pale and tense, but there may be redness of the integument resembling that of an inflammatory swelling. Itching and burning sensations usually accompany the outbreak. There may be sudden and fatal œdema of the glottis. Gastro-intestinal symptoms—nausea and vomiting, very severe abdominal pain, and diarrhœa—usually accompany the attacks. Urticaria may precede the outbreak.

The **prognosis** is good unless the larynx be involved, but recurrences are the rule.

Treatment.—A general tonic treatment is usually indicated, and indigestion and over-eating must be avoided; otherwise the treatment is unsatisfactory.

FACIAL HEMIATROPHY.

Etiology and Synonym.—This rare condition occurs more often in men than in women, and is rare after the twenty-fifth year. *Synonym:* Progressive unilateral facial atrophy.

The **pathology** of the condition is at present undefined. The disease is supposed to originate from involvement of the trophic fibres of the fifth nerve.

Symptoms.—A small area of atrophy begins in the skin, usually of the cheek or the chin, and spreads to involve half the face, being sharply limited by the median line.

The deeper structures, including the bones, participate in the atrophy, although the muscles may undergo but little change. The left side of the face is the one usually involved. Pains and peculiar sensations may accompany the earlier stages of the disease, and hemiatrophy of the tongue and of the palate may occur.

Prognosis.—The disease progresses slowly, and at any time the process may be arrested. Actual recovery does not occur, but the disease does not endanger life.

Treatment is ineffectual.

MYXŒDEMA AND CRETINISM.

Cretinism is the infantile form of myxœdema, and is due to congenital absence of the thyroid gland. The symptoms are identical with those of the adult form of myxœdema, modified by the fact that mental development and body-growth are both arrested (see Fig. 70). Cretinism may be endemic or sporadic. Symptoms may appear at birth or during the years of infancy.

Myxœdema is much more common in women than in men, and may be hereditary. The name "myxœdema" is derived from the peculiar swelling of the skin, due to the development of a myxomatous new growth. The swelling is most marked over the face and hands, but may become generally distributed. The skin and subcutaneous tissues are thickened, firm, resilient, and do not pit on pressure. The skin is dry and rough; the facies is cha-

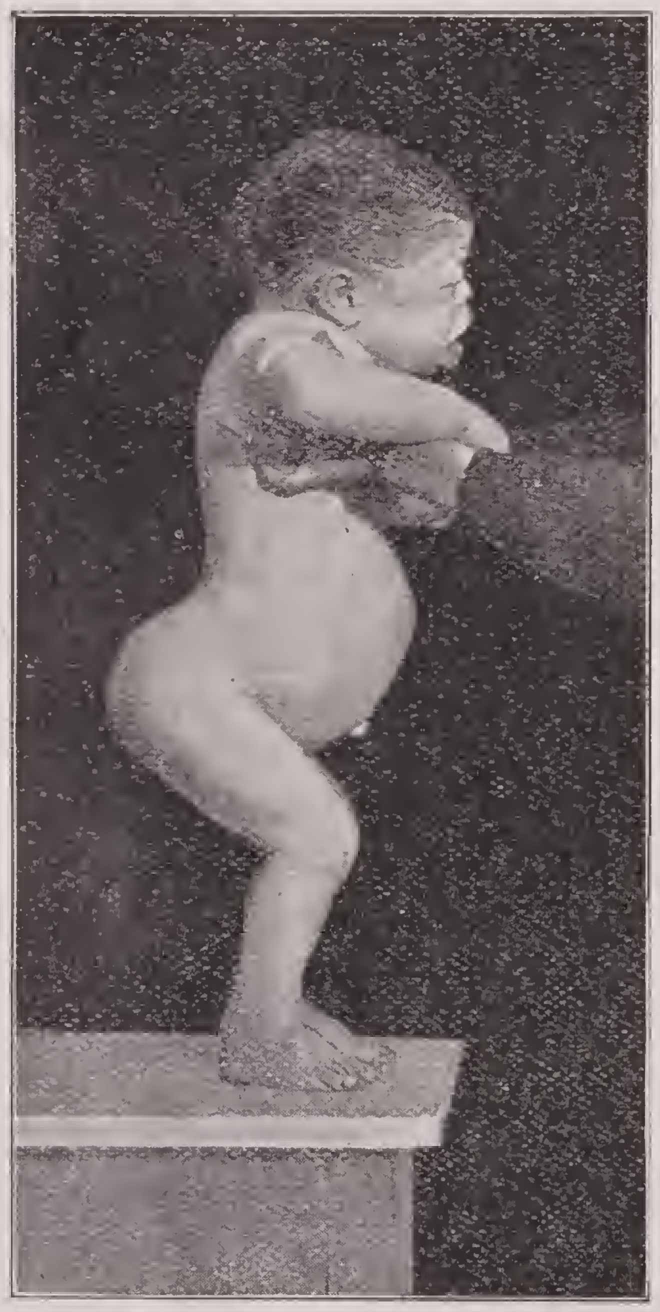

FIG. 70.—Sporadic cretinism.

racteristic; the hair becomes coarse and falls out, and a reddish patch is usually present on the cheeks. Physical and mental weakness becomes more and more marked, and may proceed to dementia. The speech is slow and monotonous. The temperature is usually subnormal. Sensory symptoms of a subjective nature, such as persistent unpleasant taste and smell, may be present. The course of the disease is slowly progressive, extending over years. Improvement under treatment, however, is to be expected.

In myxœdema the thyroid gland is usually much diminished in size, and it may become completely atrophied and converted into a fibrous mass. The most generally accepted theory is that, by reason of a failure in the function of the thyroid gland, certain injurious substances, which no longer can be rendered innocuous by the gland, collect in the body.

Operative Myxœdema ; Cachexia strumipriva.—The symptoms of myxœdema occur in animals and in men after removal of the thyroid gland. The condition follows only a certain number of complete extirpations and a smaller number of partial removals of the gland. Operative myxœdema is rare in this country.

Treatment.—It is highly important that patients with myxœdema should be kept warm and be well guarded from cold. The winters should, if possible, be spent in a warm climate. Much good can be done by hot baths, friction, and massage. The functions of the skin should be kept active by jaborandi or its alkaloid pilocarpine. The essential treatment consists in thyroid-feeding or the use of extracts from the fresh thyroid glands of sheep. From one-quarter to one-half of a gland may be given daily, or from 2 to 5 grains of the "desiccated thyroid" of any reliable maker. Care should be taken in ordering larger doses than these, as dangerous symptoms of over-dosing may ensue.

SCLERODERMA; SCLEREMA.

The causes of sclerema are unknown, although the disease is classed among the tropho-neuroses. The majority of cases occur in women of middle life. The lesion consists in the circumscribed or diffused production of connective tissue in the skin. The skin becomes hard, rigid, and adherent to the subcutaneous tissue. Pressure upon the underlying muscles may interfere with their action and may result in a certain amount of atrophy. Hyperæmia of the skin during the earlier stages may be followed by pigmentation or by atrophy of the pigment. The circumscribed form of sclerema is known as "morphœa" or as "Addison's keloid." The diffuse form may involve large portions of the body, rendering motion difficult.

The disease may develop slowly or rapidly, but when developed the lesions tend to persist for months or years. Recovery may occur, but in most instances the disease after a certain length of time becomes no longer progressive.

Massage, oil-inunctions, galvanism, avoidance of cold, and a tonic regimen constitute the essential points in the treatment.

ACROMEGALY.

Etiology.—Acromegaly occurs in both sexes during youth and middle age. The etiology of the disease is unknown.

The **pathology** is undetermined. Hyperplasia of the pituitary body is an almost constant lesion, but it is also known that similar conditions of that structure may exist without acromegaly. In some of the reported cases there has been persistence of the thymus gland.

Symptoms.—The hands and feet become enormously enlarged, owing chiefly to hypertrophy of the bones, although the muscles, skin, and subcutaneous tissues also undergo hypertrophy. The nose becomes enlarged in all dimensions, and the lower portions of the face are strikingly increased in size, so that the lower jaw usually projects far beyond the line of the upper teeth. Hypertrophy of the tongue occurs in well-marked cases. The bones of the thorax may also become enlarged, and the back may be so bowed that the chin rests upon the sternum. Sensory and vaso-motor symptoms of varied character attend the disease, and there may be persistent headache. The muscular strength is generally poor.

A condition allied to acromegaly has been described by Marie under the name of *hypertrophic pulmonary osteo-arthropathy*, this form being associated with pulmonary lesions. The condition differs from acromegaly in the following particulars: (1) The lower jaw is not enlarged; (2) the articular ends of the bones are so enlarged as to interfere somewhat with the mobility of the joint; (3) there is a peculiar bulbous deformity of the terminal phalanges.

Treatment is inoperative.

IX. DISEASES OF THE MUSCLES.

MYOSITIS.

Primary Myositis.—The few recorded cases of this disease have been characterized by swelling and tenderness of the muscles, stiffness and pain on motion, and œdema of the subcutaneous tissues. Atrophy of the muscles may result. The process may terminate fatally by involving the muscles of deglutition and of respiration. The spleen is enlarged, and an irregular erythematous eruption is usually present, the disease resembling an acute infection in these respects. Examination of the affected muscles shows marked degeneration of the fibres, with an infiltration of the interstitial tissue. The course of the disease extends over several months or years. The diagnosis from trichinosis can be positively made only by microscopic examination of the muscle-fibres. No curative treatment is known.

Acute purulent myositis, which is usually a complication of pyæmia, more rarely of other septic diseases, has occurred as a primary form in a few instances.

Progressive myositis ossificans is a rare disorder in which the muscles undergo ossification. The process may be limited to certain muscles, or may be more generally distributed, as in the well-known case of the "ossified man."

PROGRESSIVE MUSCULAR DYSTROPHY.

Etiology and Synonyms.—This disorder of development is inherited, chiefly through the mother, in three-fifths of the cases, and usually appears before the tenth year, although its onset may be deferred until the twentieth or the twenty-fifth year. The disease is four times as common in boys as in girls. The etiology of the disease is unknown. *Synonyms:* Idiopathic muscular atrophy; Pseudo-hypertrophic muscular paralysis.

Pathology.—There is an increase of connective tissue, and usually an increased deposit of fat about the muscular fibres. The muscular fibres become hypertrophied and their nuclei are increased in number. Degeneration of the fibres then ensues, and leads to atrophy, so that in advanced stages the muscles resemble masses of adipose tissue. A primary muscular atrophy may occur, with or without the development of dense connective tissue and of fat about the atrophied muscles. The spinal cord and the peripheral nerves are normal, the disorder being a primary disease of the muscles.

Symptoms.—According to the relative proportion of hypertrophy or atrophy of muscular fibres, three clinical forms are described:

1. *Pseudo-hypertrophic muscular paralysis* is the form in which increased connective tissue and fatty deposits are associated with atrophy and degeneration of the muscle-fibres. The first symptoms noted are a weakness of the muscles, usually of the calves, and an increase in their apparent bulk. The gait becomes awkward and clumsy, and there is difficulty in mounting stairs. The characteristic method of rising from the floor is by placing the hands on the knees and "climbing up the legs." The infraspinatus, the biceps, and the triceps muscles may be affected in like manner. The knee-jerk is normal; the reaction of degeneration is not obtained. Later in the disease shrinkage of the muscle-bulk occurs, with deformities. Death, when it occurs, is due to some intercurrent disease.

2. *Hypertrophic paralysis* is the form in which the muscles undergo at first a true hypertrophy, but become atrophied late in the disease. The symptoms are practically identical with those of the pseudo-hypertrophic form.

3. The *primary atrophic form* differs from the preceding forms only in the absence of a primary enlargement of muscle-bulk. A number of varieties of this form are described, according to the distribution of the lesions. Of these varieties the following are the most important:

(*a*) The *infantile form*, or the Landouzy-Déjérine type, in which the face and the shoulder-girdle are affected.

(*b*) The *juvenile form* of Erb, in which the affection first appears between the fifteenth and twentieth years, and involves the muscles of the shoulder, upper arm, gluteal region, and thigh.

Prognosis.—The course of the disease extends over years; the disease progresses steadily, and the outlook is bad.

Treatment is without influence on the disease. Galvanism and oil-inunctions with friction may be tried. The general health must be improved in every way.

PARAMYOCLONUS MULTIPLEX (MYOCLONUS MULTIPLEX).

This rare disease occurs usually in adult males, and may be induced by fright, injury, or emotion.

The **pathology** is unknown; the disease is supposed to be allied to convulsive tic.

The **symptoms** consist of sudden paroxysmal contractions, of a clonic character, of a number of muscles; the patient may be thrown from a chair or from the bed by the violent tremors. The spasms are regularly bilateral. The most characteristic spasms occur in the muscles of the trunk and hips, but the arms, legs, and face also may be involved. In exceptional cases the spasms assume a tonic form. Between attacks there may be a general muscular tremor. The body-strength is usually unimpaired, although neurasthenic or hysterical symptoms may be present.

Paramyoclonus is to be diagnosed from hysteria by the fact that the affected muscles are not physiologically allied, and therefore the spasmodic movements cannot be imitated voluntarily.

Treatment.—Chloral and hyoscine may be employed, together with a tonic treatment. Hydrotherapy seems to do good in some instances.

THOMSEN'S DISEASE; MYOTONIA CONGENITA.

This disorder, which is exceedingly rare in America, is practically always hereditary, and appears in childhood. It is unknown whether the disease is a primary disease of the muscles or a congenital defect in their innervation.

The **symptoms** consist of painless tonic spasms, lasting for a few seconds, whenever voluntary motion is attempted. Voluntary muscular contraction and relaxation are therefore slow, and the muscles seem to act stiffly. The muscles limber up, however, after repeated use, but the spasms are increased by cold or nervousness. The electrical and mechanical excitability of the muscles is increased, and the muscles themselves may undergo hypertrophy, but the bodily strength is not always good. The disease, though incurable, does not tend to shorten life.

There is no **treatment** for the disease, although Thomsen himself obtained benefit from active muscular exercise.

Congenital paramyotonia is a primary muscular affection, of a congenital and inherited character, in which tonic spasms are induced by exposure to cold. The spasms, which may occur in the extremities or in the face, may last from fifteen to twenty minutes. There is no known treatment for this disease.

X. ANIMAL PARASITES.

TREMATODES.

1. *Distoma hepaticum* (Fig. 71), or "liver-fluke," is a rare parasite 28 mm. long and 12 mm. broad. Its habitat is the small intestine, but it may enter the bile-passages and cause ascites and jaundice, with enlargement of the liver and chronic inflammation of the biliary passages.

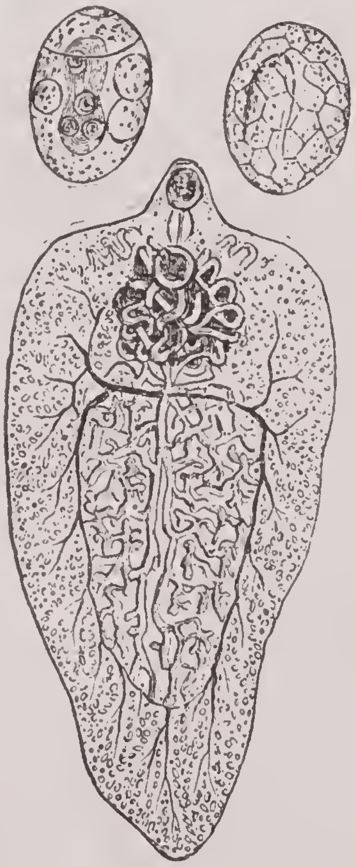

FIG. 71.—Distoma hepaticum (Von Jaksch).

Distoma lanceolatum is a smaller variety infecting cattle and sheep. *Distoma endemicum* and *distoma perniciosum* are liver-flukes endemic in Japan.

2. *Bilharzia hæmatobia*, or "blood-fluke," endemic in Egypt, Arabia, and Northern Africa, is the cause of the "endemic hæmaturia" of these countries. It is found in the abdominal veins, especially the portal, splenic, renal, and mesenteric, and is more commonly found in children. Hæmaturia, anæmia, and painful and frequent micturition are the usual symptoms. The great majority of patients recover, and the symptoms usually disappear at the time of puberty.

3. *Distoma Ringeri*, or the "bronchial fluke," gives rise to cough and hæmoptysis. The parasite is endemic in China and Japan.

NEMATODES.

Ascaris Lumbricoides.

Ascaris lumbricoïdes, or round worm, resembles in appearance the ordinary earth-worm. The female is from 12 to 16 inches in length, the male about 8 inches. This common parasite is especially frequent in Eastern countries, in women and children and in the insane. The habitat of the worm is in the small intestine, and the number varies from one to many hundreds. The parasite is usually passed by the rectum, but in rare cases the worms may wander into the bile-ducts or into the stomach, from which they may be vomited, or may pass upward to enter the larynx, bronchi, nares, or even the Eustachian tube.

The **symptoms** are usually mild and obscure. There may be vague symptoms of gastro-intestinal irritation, or reflex symptoms, such as itching of the nose, grinding of the teeth, or broken sleep. In children convulsive seizures due to worms are less common than is usually supposed.

The **diagnosis** can be made with certainty only by the actual passage of a worm.

Treatment.—Santonin may be given in doses of from ½ to 1 grain three times a day to a child of five years. Larger proportional doses may be given to adults, and the drug may advantageously be combined with small doses of calomel. Yellow vision, discolored urine, and slight delirium may attend the use of santonin in susceptible patients. When these symptoms occur the drug should at once be discontinued. Of equal service are the fluid extracts of senna and spigelia in equal parts, ʒss–j of the admixture being given three times a day until free purgation occurs.

Oxyuris Vermicularis (Pin-worm; Thread-worm).

The female worm is from 10 to 12 millimeters long; the male is one-third as long. The habitat is in the ileum and the large intestine; in these situations the parasite gives no symptoms. If the rectum be invaded, itching and burning of the anus, worse at night, occasion much distress. Excitation of the sexual organs may also result. The parasite may emigrate into the vagina and produce itching and inflammation.

The **diagnosis** is made by finding the worms in the dejections, and occasionally on the skin of the anal region.

The **treatment** consists of rectal injections of lime-water, infusion of quassia, iced salt-water, bichloride of mercury (1 : 10,000), or solution of alum (ʒj : Oj).

Anchylostoma Duodenale.

This worm (*Strongylus duodenalis*) is found in the upper portions of the small intestine, and is most common in Egypt, Italy, and Brazil. The parasite is almost unknown in the United States. The worm attaches itself by claw-like teeth to the intestinal wall, and sucks blood from the blood-vessels. The symptoms consist of digestive disorders and progressive anæmia ("Egyptian chlorosis;" "St. Gothard's disease").

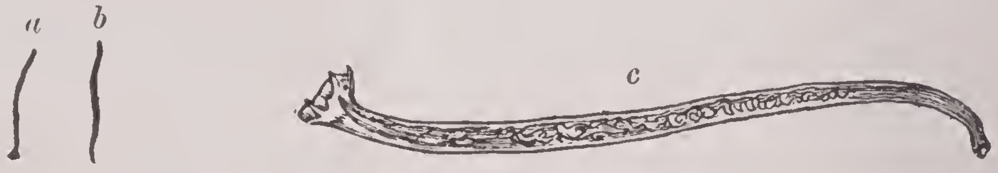

FIG. 72.—Anchylostoma duodenale (Von Jaksch): *a*, male, *b*, female, natural size; *c*, male, enlarged.

Treatment.—Thymol should be given in 30-grain doses in capsule, the dose to be repeated in two hours and followed by a brisk purgative.

Trichocephalus Dispar (Whip-worm).

This parasite, which is from ½ to 2 inches long, is characterized by a filiform anterior portion which occupies two-thirds of the entire length. The posterior portion is blunt and curved. Its habitat is in the cæcum, where the parasite is frequently found in great numbers. The worm possesses no clinical significance.

TRICHINA SPIRALIS.

When raw or imperfectly cooked ham or pork containing muscle-trichinæ is taken into the human stomach, the undeveloped trichinæ are liberated. The parasites become perfectly developed by the third day, appearing as small silvery threads barely visible to the naked eye. New-born trichinæ migrate into the muscles by the sixth day, and there assume a spiral form and become encysted, the capsule being composed of connective tissue which may be infiltrated by lime-salts. Muscle-trichinæ so encapsulated may live for years.

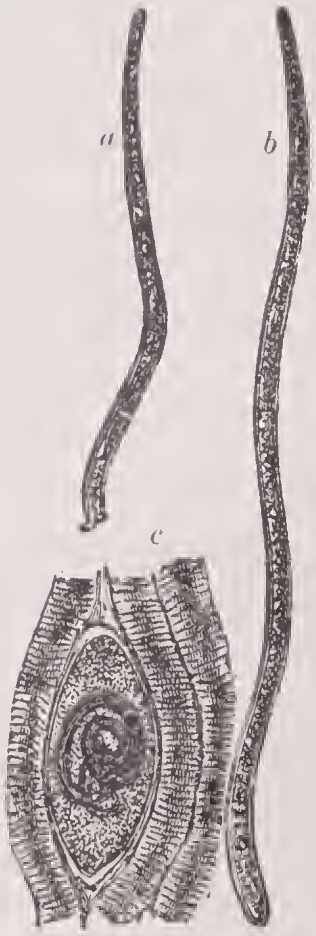

FIG. 73.—Male intestinal trichina (*a*); female intestinal trichina (*b*); muscle-trichina (*c*) (Von Jaksch).

Symptoms of trichinosis appear if trichinæ are ingested in any considerable number. The symptoms appear in two stages:

1. *Gastro-intestinal* symptoms occur one or two days after the ingestion of the infected ham. There are nausea, vomiting, abdominal pain, and serous diarrhœa. In severe cases the symptoms may resemble cholera. There may be considerable fever. In this stage very severe cases may terminate fatally.

2. *Muscular* symptoms develop in from one to two weeks. The muscles become swollen, tender, and excessively painful; the skin over the affected muscles is usually œdematous. Involvement of the respiratory muscles leads to impairment

of respiratory power, dyspnœa, and liability to bronchitis and broncho-pneumonia. Œdema of the eyelids usually appears by the seventh day, and is the most characteristic of the early symptoms. Fever of an irregular type is usually present, and profuse sweating is commonly observed. Albuminuria occurs in the majority of cases. The knee-reflexes are usually lost.

The **diagnosis** is aided by the fact that a number of individuals are usually affected at the same time. In doubtful cases a small piece of muscle may be excised under cocaïne-anæsthesia and examined.

The **duration** of the acute symptoms is from two to eight weeks. Recovery is slow and tedious.

The **prognosis** is grave, 30 per cent. of the cases terminating fatally, chiefly by pulmonary complications.

Treatment.—Prophylactic treatment consists in the governmental inspection of ham and pork and the thorough cooking of the meat.

During the gastro-intestinal stage brisk purgatives should be administered. The use of glycerin in ℥ss doses every hour has been recommended. Thymol in ʒj doses in capsule is also of service. When migration into the muscle has occurred the treatment can only be palliative.

Filaria Sanguinis Hominis.

The adult worm is from 4 to 5 inches long, and in the human subject probably lodges in some large lymphatic vessel. The female produces an enormous number of embryos from $\frac{1}{120}$ to $\frac{1}{70}$ of an inch long and of the width of a red blood-cell. The embryos enter the blood-current, and are present in the blood during the night, but disappear during the day-time. Should the patient sleep during the day and work at night, the migrations of the parasites become diurnal. It is supposed that infection occurs through the agency of mosquitoes. The parasite is most common in tropical and sub-tropical countries.

Symptoms are caused by the blocking of the lymph-channels by the adult worm or the ova. Hæmatochyluria (or chyluria) is the most common symptom. The passage of chylous urine with or without the admixture of blood is intermittent and is not inconsistent with good general health. Among the other symptoms may be mentioned lymph-scrotum, chylous hydrocele, chylous ascites, and elephantiasis.

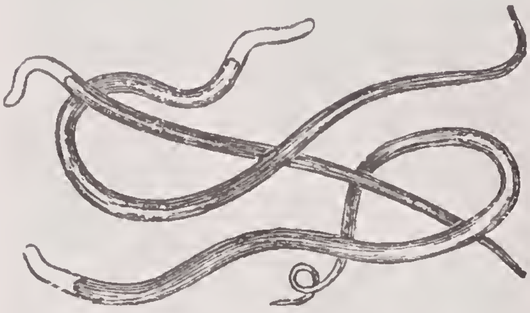
Fig. 74.—Filaria sanguinis hominis (Von Jaksch).

The **diagnosis** of parasitic chyluria from the non-parasitic form is made by the finding of the embryos in the blood drawn about midnight.

The **prognosis** is generally favorable.

Treatment.—Gallic acid (ʒj–ij doses daily) and large doses of potassium iodide have been of service. The treatment by methyl-blue has apparently been followed by good results.

CESTODES (TÆNIÆ; TAPE-WORMS).

When the eggs of the tænia enter the stomach of animals the embryos become liberated; they then migrate to other organs, where they form encysted larvæ or scolices, known as "cysticerci." A cysticercus is therefore an undeveloped tape-worm. Meat containing cysticerci is said to be "measly," and if eaten raw or imperfectly cooked the cysticerci develop into mature form within the alimentary canal of their host. Three chief varieties of tape-worm are encountered in the human subject.

1. *Tænia solium* is from 6 to 8 feet long. The head, which

is the size of the head of a pin, is marked by four suckers and a double row of hooklets. The neck is about an inch long. The joints or proglottides, which contain male and female organs of generation, become larger and more mature the further they are from the head. Tænia solium is usually single, and develops in man from eating measly pork or ham.

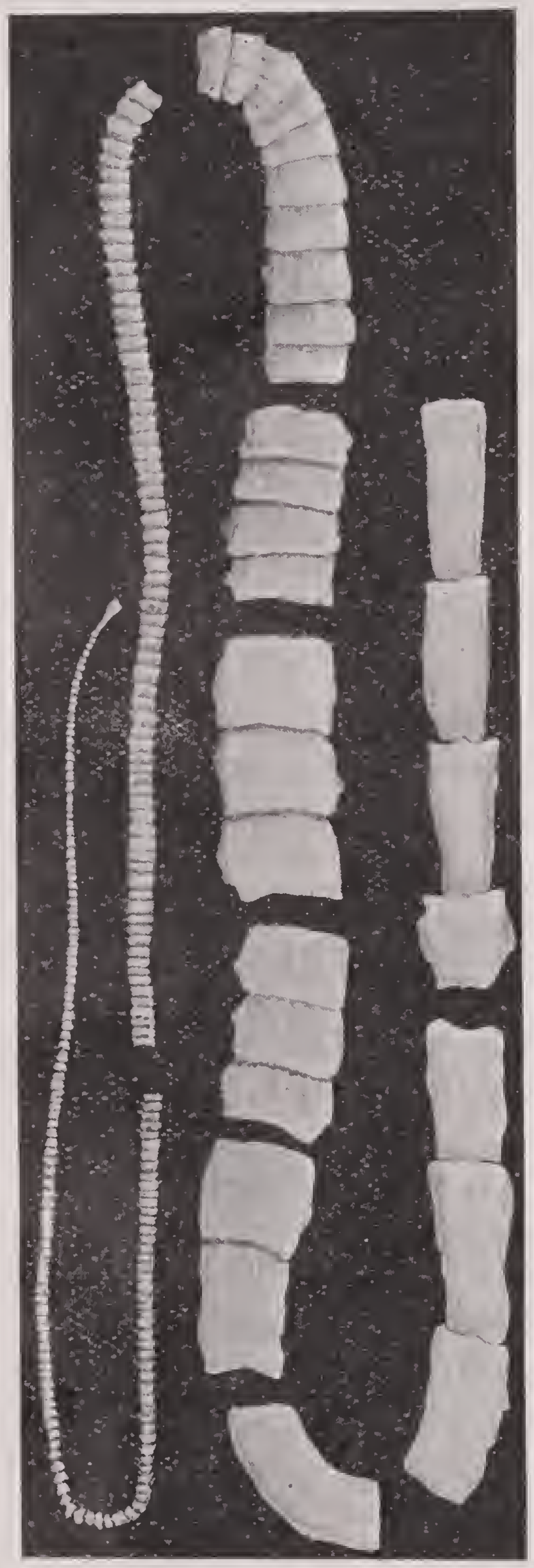

FIG. 75.—Small portions from different parts in the length of a tape-worm; natural size (Griffith).

2. *Tænia saginata* or *mediocanellata*, which is the most common form in the United States, develops from eating measly beef. The worm is longer than the tænia solium, and the sexual apparatus of the mature segments is somewhat different. The head possesses four suckers, but no hooklets.

3. *Bothriocephalus latus*, which is common in the German Baltic provinces and rare in the United States, develops from eating infected fish. The head is club-shaped, with two slit-like suckers on the side. The segments are short but broad, and the parasite frequently grows to a length of from 25 to 30 feet.

Symptoms are indefinite. There may be digestive disturbances, abdominal pain, diarrhœa, and inordinate appetite. The patient may lose flesh and strength. There may be reflex phenomena—itching of the nose, salivation, nervous vomiting, and great mental depression. The diagnosis can be made with certainty only by finding the links in the dejecta.

The growth of the bothriocephalus latus is frequently accompanied by progressive anæmia.

Treatment is successful only when the head of the worm is passed. In all cases the patient should be ordered a very light diet for two days; a saline purgative should be given on the second night, and the next morning the tæniafuge should be given on a fasting stomach and followed in two hours by a brisk purge.

Among the efficient anthelminthics recommended are fluid extract of male fern (ʒij dose), infusion of pomegranate (2 to 3 ounces of the bark in Oj of water), infusion of pumpkin-seeds, and koosso (℥ss of the dried flowers in water). Tanret's tannate of pelletierine may be given in a 5- to 10-grain dose, but the remedy is very expensive. Good results have followed ʒss doses of oil of pine-needles given in emulsion or in capsule.

ECHINOCOCCUS DISEASE.

(See *Hydatids of the Liver.*)

For the rarer forms of animal parasites the reader is referred to larger works.

INDEX.

Abscess, perinephritic, 666
  subphrenic. See *Perihepatitis, acute.*
Acetonuria, 625
Acromegaly, 904
Actinomyces, 179
Actinomycosis, etiology and pathology, 179
  prognosis, 180
  symptoms and treatment, 180
Addison's disease, 731
    pathology, 731
    symptoms, 732
    treatment, 733
Albuminuria, 614
  tests for, 616
Amyloid degeneration, intestinal ulcers due to, 518
Anæmia, 712
  pernicious, 718
    pathology, 718
    symptoms, 720
    treatment, 721
  secondary, 712
    pathology and symptomatology, 713
    treatment 715
Aneurysm, 281
  of abdominal aorta, 290, 291
  of thoracic aorta, 283
      diagnosis, 287
      physical signs, 286
      prognosis, 288
      treatment, 289
Angina pectoris, 266
    prognosis and treatment, 268
    symptoms, 267
  pseudo-, 269
Animal parasites, 910
Ankylostoma duodenale, 912
Anthrax, 163
  definition and synonyms, 163
Anthrax, etiology, 163
  symptoms, 163
  treatment, 165
    of the disease, 168
    preventive inoculations, 167
Aortic regurgitation, 234
  stenosis, 239
Apoplexy, 749
  pulmonary, 323
Appendicitis, 505
  acute catarrhal, 506
      course of the disease, 508
      pathology and symptoms, 506
      physical examination, 507
      symptoms, 506
  acute suppurative, 508
      course of the disease, 511
      pathology, 508
      physical signs, 510
      symptoms, 509
  chronic, 515
  gangrenous, 513
    prognosis, 513
    treatment, 514
  ulcerative, 508
Arteries, cerebral, aneurysm of, 758
    embolism of, 755
      etiology and pathology, 755
      diagnosis, 757
      symptoms, 756
      treatment, 757
    hemorrhage and embolism of, differential diagnosis, 757
    thrombosis of, 758
  diseases of, 277
Arterio-sclerosis, 277
  pathology, 278
  physical signs, 280
  symptoms, 279
  treatment, 280
Arteritis, syphilitic, 281

Arthritis deformans, 686
    pathology and symptoms, 687
    prognosis and treatment, 689
  rheumatoid. See *Arthritis deformans.*
Ascarides lumbricoides, 901
Ascites, 553
  tubercular, 549
Asphyxia, local, 900
Asthma, 313
  causes, exciting, 314
    predisposing, 313
  definition and etiology, 313
  pathology and symptoms, 314
  physical signs, 315
  prognosis and treatment, 316
  thymic, 292
Asthma-crystals, 316
Ataxia, hereditary, 821
  locomotor, 813
    Charcot's knee in, 818
Atheroma, 225. See *Arterio-sclerosis.*
Atrophy, acute yellow. See *Hepatitis, parenchymatous, acute.*
  cerebral, in children, 778
      etiology and symptoms, 778
      treatment, 780
  facial, progressive unilateral. See *Hemiatrophy, facial.*
  progressive muscular, 804
      diagnosis, 806
      pathology, 804
      prognosis, 807
      symptoms, 805
      treatment, 807
Auditory nerve, diseases of, 838

BACILLUS, comma, 127
  Klebs-Loeffler. See Plate 10.
  lepræ, 173
  tetani, 169
  typhosus, 18
Basedow's disease. See *Exophthalmic goitre.*
Bell's mania. See *Delirium, acute.*
  palsy, 835
Beri-beri, 854
Bilharzia hæmatobia, 910
Blood, diseases of, 712
  vomiting of, in ulcer of the stomach, 461
Blood-fluke, 910
Bothriocephalus latus, 916
Bowels, changes in, in cancer of the intestine, 523
  hemorrhages from, in typhoid fever, 28
Brachycardia, 264
Bradycardia, 264
Brain, abscess of, 769
    pathology and symptoms, 770
    treatment, 771
  blood-vessels of, anæmia of, 748
    congestion of, 747
    œdema of, 749
  cortical areas of, 764
  gummata of, 786
  membranes of, diseases of, 736
  subcortical areas of, 768
  syphilis of, 785
    symptoms, 787
    treatment, 788
  tumors of, 772
    pathology and symptoms, 773
    treatment, 777
Brain-substance, diseases of, 764
Brand, method of, in treatment of typhoid fever, 42
Breast-pang. See *Angina pectoris.*
Bright's disease, acute. See *Nephritis.*
    chronic, 635
Bright's hypertrophy of the heart, high tension in, 205
Brodie's joint, 893
Bronchi, diseases of, 302
Bronchial fluke, 910
Bronchiectasis, 310
Bronchitis, capillary. See *Broncho-pneumonia.*
  catarrhal, acute, 302
    chronic, 308
  croupous, acute, 306
    chronic, 310
  diphtheritic. See *Bronchitis, acute croupous.*
  membranous, fibrinous, and plastic. See *Bronchitis, chronic croupous.*
Broncho-pneumonia, 341
  cerebral cases, 346
  course and termination, 346
  in adults, 348
    prognosis and treatment, 350
  pathology, 342
  physical signs, 346
  symptoms, 344

CACHEXIA, malarial, 160
treatment, 162
strumipriva, 903
Caisson-disease, 796
Calculi, biliary. See *Cholelithiasis.*
renal, 659
Calomel fumigation in treatment of diphtheria, 114
Caput coli, cancer of, 524
Cardiac affections complicating rheumatism, 670
Cardialgia, 447
Cerebral complications of rheumatism, 671
Cestodes, 915
Charbon. See *Anthrax.*
Charcot-Leyden's asthma-crystals, 316
Charcot's joint in locomotor ataxia, 818
Chicken-pox. See *Varicella.*
Chloro-anæmia. See *Chlorosis.*
Chloroform inhalations in treatment of hydrophobia, 168
Chlorosis, 716
Cholecystitis, suppurative, 599
Cholelithiasis, 594
symptoms, 596
treatment, 601
Cholera, 126
bacillus of, 127
complications and sequelæ, 131
contagiousness of, 128
definition and synonym, 126
etiology, 127
incubation period, 128
infantum, 492
in meat-fed children, 495
morbus, 496
pathology, 128
prognosis, 131
stage of collapse, 130
stage of preliminary diarrhœa, 129
stage of purging, 129
stage of reaction, 130
symptoms, 128
treatment, medicinal, 132
prophylactic, 131
warm or tepid stage, 130
Cholerine, 131
Chorea, 873
habit-, 879
Huntingdon's, 879
insaniens, 874
symptoms, 874
Chorea, treatment, 877
Chyluria, 624
Circulatory system, diseases of, 183
Cirrhosis, biliary, 570
fatty, 571
Coccygodynia, 858
Colic, biliary, pulse in, 597
mucous. See *Colitis, mucous.*
Colitis, mucous, 487
Compression-myelitis, 829
Congestions, venous, acute, in dilatation of the heart, 208
chronic, in dilatation of the heart, 209
Constitutional diseases, 667
Consumption, chronic. See *Phthisis, chronic pulmonary.*
galloping. See *Phthisis, acute pulmonary.*
Convulsions, infantile, 860
causes, 860
Cor adiposum, 254
Coup de soleil, 896
Cramp, writer's, 884
Cranial bones, syphilis of, 786
nerves, diseases of, 831
Creosote in treatment of tuberculosis, 398
Cretinism, 902
Croup, spasmodic, 293
Curschmann's spirals, 315
Cyst, echinococcus, 583
Cysts, pancreatic, 609

DEGENERATION, amyloid, following tertiary syphilis, 142
reaction of, 800
Delirium, acute, 871
tremens, 898
Dementia, paretic. See *Paresis, general.*
Dengue, 94
convalescence from, 95
etiology, 94
pathology and symptoms, 94
prognosis and treatment, 95
Diabetes insipidus, 697
treatment, 698
mellitus, 689
diet in, 695
etiology and pathology, 690
symptoms, 691
treatment, 695

Diarrhœa, acute dyspeptic, in children, 490
  chronic. See *Enteritis, chronic catarrhal.*
  in intestinal ulceration, 520
  morning, 480
  summer, in children, 490
Diarrhœal diseases of children, 489
    cholera infantum, 492
    diarrhœa, acute dyspeptic, 490
    entero-colitis, acute, 490
Digestive system, diseases of, 432
Diphtheria, 102
  antitoxine of, 115
  complicating lesions, 105
  complications and sequelæ, 109
  definition, 102
  essential lesion, 104
  etiology, 102
  immunity from, 103
  incubation period, 105
  laryngeal, 108
  local, 105
  method of infection, 103
  mortality, 111
  nasal, 108
  pathology, 104
  pharyngeal, 107
  prognosis, 111
  serum-therapy, 115
  symptoms, 105
  tonsillar, 106
  treatment, 111
    general, 112
    local, 112
    prophylactic, 111
Diplococcus pneumoniæ of Fränkel, 325
Diseases, constitutional, 667
Distoma hepaticum, 910
  lanceolatum, 910
  Ringeri, 910
Dropsy, abdominal, 553
Duchenne's paralysis, 803
Duodenum, cancer of, 525
  round ulcer of, 516
  ulceration of, after burns, 517
Dura mater, cerebral, diseases of, 736
    hæmatoma of. See *Pachymeningitis, chronic internal.*
  spinal, diseases of, 788
Dysentery, 497
  acute, treatment, 503
Dysentery, acute, treatment: amœbic form, 504
    catarrhal form, 503
    croupous form, 504
  catarrhal, acute, 498
    treatment, 503
  chronic, 504, 505
  croupous, acute, 501
    treatment, 504
  tropical or amœbic, 499
    treatment, 504
Dyspepsia, chronic. See *Gastritis, chronic catarrhal.*
  nervous, 475
Dystrophy, progressive muscular, 906

ECHINOCOCCUS, 583
  cyst. See *Liver, hydatid of.*
  cysts of the lung, 368
  disease, 917
Eclampsia, infantile, 860
Effusion in pleurisy, mechanical effects of, 404
Emphysema, interlobular, 355
  vesicular, 356
    pathology, 357
    physical signs, 360
    prognosis, 360
    symptoms, 358
    treatment, 361
    variations, 359
Empyema, 411
Encephalitis, suppurative, 769
Endarteritis, cerebral, 761
    illustrative case, 762
    pathology and symptoms, 761
    prognosis and treatment, 763
  chronic. See *Arterio-sclerosis.*
  syphilitic, 786
Endocarditis, acute, 212
    course of the disease, 217
    effects of the lesion, 215
    etiology, 213
    pathology, 214
    physical signs, 216
    prognosis, 217
    symptoms, 215
    treatment, 218
  chronic, 223
    treatment, 243
  complicating lobar pneumonia, 337
  malignant, 219
    diagnosis, 222

Endocarditis, malignant, duration, 222
    etiology, 219
    lesion, 219
    prognosis, 223
    symptoms, 220
    treatment, 223
Enteritis, catarrhal, acute, 481
      etiology, 481
      pathology and symptoms, 482
      prognosis and treatment, 483
  catarrhal, chronic, 483
      etiology, 483
      pathology and symptoms, 484
      prognosis and treatment, 485
  phlegmonous, 486
  pseudo-membranous, 486
Entero-colitis, acute, in children, 490
Enterorrhagia, 531
Epilepsy, 862
  and hystero-epilepsy, differential diagnosis, 867
  causes, 862
  diagnosis, 866
  Jacksonian, 866
  nocturnal, 865
  pathology, 863
  procursive, 863
  prognosis and treatment, 867
  symptoms, 863
Epithelioma of the stomach, 465
Erysipelas, 117
  complications, 120
  definition, 117
  etiology, 117
  metastatic, 119
  migrans, 119
  pathology, 117
  prognosis, 120
  streptococci of, effect on tumor-cells, 120
  symptoms, general, 118
    local, 118
  traumatic and idiopathic forms, 117
  treatment, general, 120
    local, 121
    prophylactic, 120
Eshbach's test for albumin in urine, 617
Expiration, forced, arrangement of bottles for, 415

FACIAL NERVE, spasm of, 838
Farcy. See *Glanders.*
Fecal accumulation, 533
Fever, breakbone. See *Dengue.*
  cerebro-spinal. See *Meningitis, epidemic cerebro-spinal.*
  fermentation-, 124
  in abscess of the liver, 575
  malarial. See *Malarial fever.*
  relapsing. See *Relapsing fever.*
  rheumatic, 667
  scarlet. See *Scarlet fever.*
  thermic, 896
    treatment, 897
  typhoid. See *Typhoid fever.*
  yellow. See *Yellow fever.*
Fibro-serous membranes, inflammation of, in acute articular rheumatism, 669
Fifth nerve, diseases of, 833
Filaria sanguinis hominis, 914
Foot-drop, 851
Foramen ovale, patency of, congenital, 275
Fourth nerve, diseases of, 833
Friedreich's ataxia. See *Ataxia, hereditary.*

GALL-BLADDER, calculi in, 594
Gall-ducts, cancer of, 602
Gall-stones. See *Cholelithiasis.*
Gangrene, local or symmetrical, 900
Garland's S-curve, 406
Gastralgia, 477
Gastrectasis, 452
Gastric juice, hyperacidity of, 476
    hypersecretion of, 476
Gastritis, catarrhal, acute, 440
      etiology and synonyms, 440
      from ptomaïne-poisoning, 441
      pathology and symptoms, 440
      treatment, 441
    chronic, 445
      etiology and pathology, 445
      prognosis, 449
      treatment, 449
        dietetic and hygienic, 449
        medicinal, 450
  croupous, acute, 443
  diphtheritic. See *Gastritis, acute croupous.*
  gastric, acute. See *Gastritis, acute catarrhal.*
  membranous. See *Gastritis, acute croupous.*

Gastritis, mycotic and parasitic, 444
  phlegmonous. See *Gastritis, acute suppurative.*
  purulent. See *Gastritis, acute suppurative.*
  sclerotic, 446, 449
  simple, 446, 449
  suppurative, acute, 444
  toxic, 442
    symptoms and treatment, 443
Gastro-duodenitis. See *Jaundice, catarrhal.*
Gastrodynia, 477
Gastrorrhagia. See *Stomach, hemorrhage from.*
Glanders, 176
  acute, 177
  chronic, 178
  definition and synonym, 176
  etiology, 176
  lesion, 177
  symptoms, 177
  treatment, 178
Glosso-pharyngeal nerve, diseases of, 840
Glottis, œdema of. See *Laryngitis, œdematous.*
Glycosuria, 625, 696
Goitre, exophthalmic, 271
    prognosis, 273
    treatment, 274
Gout, 678
  associated lesions of, 680
  etiology, 678
  pathology, 680
  rheumatic. See *Arthritis deformans.*
  symptoms, 681
  treatment, 684
Grand mal, 863
    auræ in, 863
Graves' disease. See *Exophthalmic goitre.*
Green sickness. See *Chlorosis.*
"Griffin-claw," 805
Gummata in the lung, 365
  in the myocardium, 253
  of syphilis, 141
  of the brain, 786

HÆMATEMESIS. See *Stomach, hemorrhage from.*
Hæmatoma, mediastinal, 431
Hæmaturia, 617
Hæmoglobinuria, 619
  paroxysmal, 620
  toxic, 619
Hæmopericardium, 197
Hæmophilia, 709
  symptoms, 710
  treatment, 711
Hæmoptysis, 320
  and hæmaturia, differential diagnosis, 474
  arthritic, 321
Hæmothorax, 426
Headache, sick. See *Migraine.*
Heart, aneurysm of, 259
    symptoms, prognosis, and treatment, 260
  dilatation of, 206
    physical signs, 211
    symptoms, 207
  diseases of, 198
  fatty, 254
  hypertrophy of, 202
    symptoms, 203
  intermittent action of, 263
  malformations of, congenital, 275
      symptoms and treatment, 277
  neuralgia of. See *Angina pectoris.*
  neuroses of, 261
  palpitation of, 261, 262
  rupture of, 260
    symptoms, prognosis, and treatment, 261
Heartburn, 447
Heart-muscle, poisoning of, 255
  weakness of, 198
Heat-exhaustion, 895
Heat-stroke, 896
Heberden's nodes, 687
Heller's test for albumin in urine, 616
    for blood in urine, 618
Hemiatrophy, facial, 901
Hemicrania. See *Migraine.*
Hemiplegia, infantile, 778
Hemorrhage between cerebral dura mater and pia mater, 746
  between cerebral dura mater and the bones of the skull, 745
  broncho-pulmonary, 320
  cerebral, 749
    etiology, 749
    pathology, 750
    prognosis, 754

Hemorrhage, cerebral, symptoms, 751
    treatment, 754
  from the bowels in typhoid fever, 29
  in cancer of the intestine, 523
  in intestinal ulceration, 520
  in jaundice, 591
  into the spinal cord, 795
  into the spinal membranes, 793
  meningeal, 745
  pulmonary, 320
  renal, 617
Hemorrhages in cancer of the stomach, 468
  in chronic pulmonary phthisis, 390
Hemorrhagic infarct, 323
Henoch's disease, 708
Hepatitis, interstitial, chronic. See *Liver, cirrhosis of.*
  parenchymatous, acute, 562
  suppurative, 573
Hodgkin's disease. See *Pseudo-leukæmia.*
Huntingdon's chorea, 879
Hutchinson's teeth, 147
Hydrocephalus, chronic, 784
    acquired, 785
    congenital, 784
    treatment, 785
Hydronephrosis, 657
Hydropericardium, 196
Hydroperitoneum, 553
Hydrophobia, 165
  etiology, 165
  incubation period, 166
  pathology, 166
  prognosis, 167
  symptoms, 166
  treatment, 167
Hydro-pneumothorax, 420
Hydrothorax, 425
Hyperacidity of gastric secretions, 476
Hyperpyrexia in rheumatism, 671
Hypersecretion of gastric juice, 476
Hypoglossal nerve, diseases of, 846
Hysteria, 887
  symptoms, 888
    circulatory, 892
    digestive, 891
    joint, 893
    mental, 893
    motor, 888
    respiratory, 892
    sensory, 890
Hysteria, symptoms: special senses, 891
    temperature, 893
    urinary, 892
  treatment, 894

ICTERUS. See *Jaundice.*
Impaction, fecal, complete, 534
    incomplete, 535
Incompetency, mitral, 226
Indicanuria, 624
Indigestion, acute, 441
Infection, septic, 124
Infectious diseases, 17
Influenza, epidemic, 89
    bacillus of, 89
    complications and sequelæ, 91
      pneumonia, 91
    convalescence from, 92
      treatment during, 93
    course, 91
    definition and synonym, 89
    duration, 92
    etiology, 89
    lesions, 89
      complicating, 89
    prognosis, 92
    symptoms, 90
      catarrhal, 90
      gastro-intestinal, 91
      nervous, 90
      of infection, 90
    treatment, 92
Inoculations, preventive, in treatment of hydrophobia, 167
Insolation, 896
Intestinal obstruction from foreign bodies, 527
    from strictures and tumors, 527
Intestine, amyloid degeneration of, 536
  cancer of, 521
    pathology, 522
  hemorrhage from, 531
    symptoms and treatment, 532
  non-cancerous tumors of, 525
  ulceration of, 516
    symptoms, 520
    treatment, 521
    varieties, 516
Intestines, diseases of, 480
Intoxication, septic, 124

JAUNDICE, 589
  bowels in, 591

Jaundice, catarrhal, 592
  febrile, acute. See *Weil's disease.*
  malignant. See *Hepatitis, parenchymatous, acute.*
  temperature in, 592
  urine in, 591

Kak-ke, 854
Kidney, amyloid, 647
  congenital cystic, 656
  cysts of, 655
  fatty. See *Kidneys, chronic degeneration of.*
  floating. See *Kidney, movable.*
  movable, 612
  palpable. See *Kidney, movable.*
  suppurative disease of, 650
      etiology, 650
      pathology and symptoms, 651
      treatment, 653
  surgical, 650
  tubercular diseases of, 648
  tuberculosis of, 648
  tumors of, 653
    symptoms, 654
    treatment, 655
  waxy degeneration of, 647
Kidneys, congestion of, acute, 625
    chronic, 626
  degeneration of, acute, 627
    chronic, 628
  diseases of, 612
  malformations of, congenital, 612
Knee-jerk, loss of, in locomotor ataxia, 815
Koch, bacillus of, 127
Koch's lymph in treatment of tuberculosis, 398

La grippe. See *Influenza, epidemic.*
Landry's paralysis. See *Paralysis, acute ascending.*
Laryngismus stridulus, 292
Laryngitis, catarrhal, acute, 294
    chronic, 296
  membranous, 297
  œdematous, 301
  spasmodic, 293
  syphilitic, 300
  tubercular, 298
Larynx, diseases of, 292
  spasm of, 292
Lead-paralysis, 853
Leontiasis, 174
Leprosy, 173
  anæsthetic, 174
  bacillus of, 173
  definition, 173
  etiology, 173
  pathology, 173
  prognosis, 176
  symptoms, 174
  treatment, 176
  tubercular, 174
Leptomeningitis, acute, 790
  chronic, 792
Leucocythæmia, 722
Leucocytosis, 722
Leukæmia, 722
  blood-changes in, 723
  diagnosis and prognosis, 726
  etiology and pathology, 722
  lymphatic, 724
  pseudo-, 727
  spleen in, 724
  symptoms, 725
  treatment, 726
Lipuria, 625
Lithæmia, 622
Liver, abscess of, 573
    course, 577
    diagnosis, 578
    pathology, 574
    physical examination, 576
    symptoms, 575
    treatment, 579
  amyloid, 588
  blood-vessels of, diseases of, 603
  capsule of, diseases of, 560
  circulatory disturbances of, 559
  cirrhosis of, 565
    atrophic, 565
    fibrous, 565
    hypertrophic, 570
    syphilitic, 571
  congestion of, passive, 559
  diseases of, 556
    tubercular, 579
  fatty, 586
  functional disturbances of, 556
  gummata of, 572
  hydatid of, 583
  lardaceous. See *Liver, amyloid.*
  new growths of, 579
  "nutmeg," 559
  size of, in cirrhosis, 598

Liver, waxy. See *Liver, amyloid.*
Liver-fluke, 910
Localization, cerebral, 764
Lock-jaw, 834. See *Tetanus.*
Locomotor ataxia, 813
    diagnosis, differential, 819
    localization of lesion in, 814
    pain in, 815
    pathology, 813
    perverted sensations in, 815
    prognosis, 819
    symptoms, 815
    treatment, 820
    trophic changes in, 818
    visceral crises in, 817
Lung, abscess of, 364
  echinococcus cysts of, 368
  gangrene of, 362
    symptoms, 363
    treatment, 364
  gummata in, 365
  new growths of, 366
  syphilis of, 365
  tubercular inflammations of, 369
      conditions favorable to infection, 370
      modes of infection, 369
      secondary inflammatory processes, 373
      structure of tubercle, 371
      symptoms, 373
  white hepatization of, 365
Lungs, circulatory disturbances of, 318
  congestion of, 318
  diseases of, 318
  œdema of, 319
Lymphadenitis, 427
Lymph-glands, tuberculosis of, 733
    pathology and symptoms, 734
    treatment, 735

MALARIAL FEVER, 152
    clinical forms, 155
        intermittent fever, 155
        malarial cachexia, 160
        pernicious intermittent fever, 157
        pernicious remittent fever, 159
        remittent fever, 158
    conditions favoring infection, 153
    definition, 152
    diagnosis, 160
    etiology, 152
Malarial fever, pathology, 154
    prognosis, 160
    treatment, 161
      quinine in, 161
Mania à potu, 898
Measles, 76
  complications and sequelæ, 79
  contagion of, 77
  definition and synonyms, 76
  German. See *Rubella.*
  pathology, 77
  prognosis, 80
  symptoms, 77
    stage of eruption, 78
    stage of invasion, 77
  treatment, 80
  variations in course of, 79
Mediastinum, abscess of, 430
  diseases of, 427
  emphysema of, 430
  tumors of, 428
Melæna neonatorum, 473
Ménière's disease, 839
Meningitis, chronic, 744
  complicating lobar pneumonia, 337
  epidemic cerebro-spinal, 95
      anomalous cases, 99
      complications, 100
      definition and synonym, 95
      etiology, 95
      pathology, 96
      prognosis, 101
      sequelæ, 100
      symptoms, 97
      treatment, 101
  non-tubercular, acute, 741
      etiology, 741
      pathology, 742
      symptoms, 743
      treatment, 744
  syphilitic, 744, 786
  tubercular, 739
    and simple, differential diagnosis, 743
Merycismus, 480
Migraine, 882
Milk sickness, 181
Milzbrand. See *Anthrax.*
Mitral incompetency, 226
  stenosis, 230
Morbus maculosus, 706
Morphine hypodermically in treatment of hydrophobia, 168

Mumps. See *Parotitis, epidemic.*
Muscles, diseases of, 906
  laryngeal, spasm of, 842
Mycosis intestinalis, 164
Myelitis, acute and chronic, 822
      pathology, 822
      prognosis, 826
      symptoms, 823
      treatment, 826
  compression-, 829
Myocarditis, acute, 249, 250
  chronic, 251, 252
  diffuse fibroid, 252
  syphilitic, 252
Myocardium, degeneration of, 253
    pathology and symptoms, 256
    physical signs, 258
    prognosis and treatment, 258
  diseases of, 249
  gummata in, 253
Myoclonus multiplex, 908
Myositis, 906
  ossificans, progressive, 906
Myotonia congenita, 909
Myxœdema, 902
  operative, 903
  treatment, 903

NECROSIS, anæmic, of myocardium, 253
Nematodes, 911
Nephritis, chronic, 635
  diffuse, acute, 632
    chronic, with exudation, 635
      course, 638
      etiology and synonyms, 635
      pathology, 636
      prognosis, 639
      symptoms, 637
      treatment, 639
    without exudation, 639
      cause, 644
      etiology and synonyms, 639
      lesions, complicating, 641
      pathology, 640
      prognosis, 645
      symptoms, 641
      treatment, 645
  exudative, acute, 629
  parenchymatous. See *Kidneys, degeneration of, acute.*
Nephrolithiasis, 659
  etiology, 659
Nephrolithiasis, pathology, 660
  symptoms, 660
  treatment, 664
Nephroptosis. See *Kidney, movable.*
Nerve, auditory, diseases of, 838
  facial, diseases of, 835
    spasm of, 838
  fifth, diseases of, 833
    neuralgia of, 834
  fourth, diseases of, 833
  glosso-pharyngeal, diseases of, 840
  hypoglossal, diseases of, 846
  olfactory, diseases of, 831
  optic, diseases of, 831
  pneumogastric, diseases of, 840
  seventh, diseases of, 835
  sixth, diseases of, 834
  spinal accessory, diseases of, 844
  third, diseases of, 832
Nerve-fibres, degeneration of, 849
  normal, 849
Nerve-trunks, tumors of, 855
Nerves, cranial, diseases of, 831
  peripheral, diseases of, 847
Nervous diseases, general, 860
  system, diseases of, 736
Neuralgia, 856
  cervico-brachial, 857
  cervico-occipital, 857
  clinical varieties, 857
  intercostal, 858
  lumbar, 858
  prognosis and treatment, 859
  symptoms, 856
  trifacial, 857
Neurasthenia, 885
  treatment, 887
Neuritis, alcoholic, 851
  coal-gas, 854
  localized, 847
    interstitial, 848
    parenchymatous, 848
    prognosis and treatment, 850
  multiple, 850
    symptoms, 850
    treatment, 854
  optic, in brain-tumors, 774
  post-febrile, 852
  symptoms, 849
Neuroma, plexiform, 855
Neuromata, 855
Neuroses, occupation-, 884
Nodules, rheumatic, 672

OBSTRUCTION, intestinal, 525
    diagnosis, 528
    etiology and pathology, 525
    prognosis and treatment, 530
    symptoms, 528
Occupation-neuroses, 884
Œdema, acute circumscribed, 900
  angio-neurotic, 900
  malignant. See *Anthrax.*
Œsophagismus, 437
Œsophagitis, acute, 432
  chronic, 433
Œsophagus, dilatations and diverticula of, 438
  diseases of, 432
  paralysis of, 439
  rupture of, 439
  stenosis of, 433
    spasmodic, 437
  stricture of, 433
    cancerous, 435
    cicatricial, 435
  varix of, 439
Olfactory nerve, diseases of, 831
Ophthalmoplegia externa, 810
  interna, 810
Optic nerve, diseases of, 831
  neuritis in brain-tumors, 774
Osteoarthropathy, hypertrophic pulmonary, 905
Oxaluria, 624
Oxyuris vermicularis, 911

PACHYMENINGITIS, external, acute, 736
  internal, acute, 736
    chronic, 737
  syphilitic, 738
Palsy, Bell's, 835
  shaking. See *Paralysis agitans.*
  wasting. See *Atrophy, progressive muscular.*
Pancreas, cancer of, 610
  diseases of, 605
  hemorrhage into, 605
Pancreatic cysts, 609
Pancreatitis, chronic, 608
  gangrenous, 607
  hemorrhagic, acute, 606
  suppurative, 608
Paralysis, acute ascending, 827
  agitans, 869
  arsenical, 853
Paralysis, atrophic spinal, 797
  Brown-Séquard's, 830
  bulbar, 808
    acute, 808
    chronic, 808
  diver's, 796
  facial, 835, 837
  glosso-labio-laryngeal. See *Paralysis, bulbar.*
  infantile. See *Poliomyelitis, anterior, in children.*
  Landry's. See *Paralysis, acute ascending.*
  laryngeal, 841
  lead-, 853
  muscular, hypertrophic, 908
    primary atrophic, 908
    pseudo-hypertrophic, 907
  syphilitic, 787
Paramyoclonus multiplex, 908
Paramyotonia, congenital, 909
Paraplegia, ataxic, 821
  spastic, of infants, 812
Parasites, animal, 910
Paresis, general, 782
Parkinson's disease. See *Paralysis agitans.*
Parotitis, epidemic, 83
    complications and sequelæ, 84
    definition and synonym, 83
    etiology, 83
    lesion, 83
    symptoms, 83
    treatment, 84
Peliosis rheumatica, 705
Peptonuria, 621
Pericarditis, 183
  and dilatation, differential diagnosis, 190
  cancerous, 196
  chronic adhesive, 193
  clinical types, 189
  complicating lobar pneumonia, 337
  diagnosis, 190
  etiology, 183
  lesion, 184
  physical signs, 187
  prognosis, 191
  purulent, 192, 193
    pathology and symptoms, 192
    prognosis and treatment, 193
  symptoms, 186
  terminations, 185

Pericarditis, treatment, 191
  tubercular, 195, 196
Pericardium, diseases of, 183
Periencephalitis, acute. See *Delirium, acute.*
Perihepatitis, acute, 560
  chronic fibrinous, 561
  syphilitic, 572
Perinephritic abscess, 666
Peristaltic unrest, 479
Peritoneum, cancer of, 551
  diseases of, 536
  tubercular inflammations of, 547
  tuberculosis of, acute, 547
Peritonitis, acute, 536
    bacteriology, 537
    etiology, 536
    varieties, 537
  chronic, 545
  circumscribed, acute, 542
    treatment, 544
  diffuse, acute, 538
      diagnosis, 541
      pathology, 538
      symptoms, 539
      treatment, 543
  hemorrhagic, chronic, 547
  treatment, 543
  tubercular, acute, 548
    chronic, 549
Pertussis. See *Whooping-cough.*
Petit mal, 865
Pharyngitis complicating rheumatism, 673
Phosphaturia, 622
Phthisis florida. See *Phthisis, pulmonary, acute.*
  pulmonary, acute, 328
    diagnosis, differential, 384
    pathology, 382
  pulmonary, chronic, 386
      complications, 394
      pathology, 386
      physical signs, 393
      symptoms, 388
      treatment, 395
Pia mater, cerebral, diseases of, 739
    spinal, diseases of, 790
Pin-worms, 911
Plasmodium malariæ, 152
Pleura, diseases of, 400
  new growths of, 424
Pleurisy, chronic, 416
Pleurisy, chronic, course and diagnosis, 418
    symptoms and physical signs, 417
    treatment, 418
  complicating lobar pneumonia, 336
  effusion in, mechanical effects of, 404
  fibrinous or dry, 400
  plastic, 400
    pathology and symptoms, 401
    physical signs, 402
    prognosis and treatment, 403
  purulent, 411
    course, 414
    pathology, 412
    prognosis, 415
    symptoms, 413
    treatment, 415
  tubercular, 418
  with effusion, 403
      course, 409
      sequelæ, 409
      symptoms, 404
      treatment, 410
Pneumogastric nerve, diseases of, 840
Pneumonia, broncho-, 341
  catarrhal. See *Broncho-pneumonia.*
  complicating epidemic influenza, 91
  interstitial, 352
    prognosis and treatment, 355
    symptoms, 354
  lobar, 324
    complications, 336
    etiology, 325
    pathology, 326
    physical signs, 333
    symptoms, 328
      cerebral, 332
    treatment, 339
  lobular. See *Broncho-pneumonia.*
  prognosis, 338
  syphilitic fibroid, 366
Pneumopericardium, 197, 198
Pneumothorax, 420
  diagnosis, 423
  symptoms, 421
  treatment, 424
Podagra, 678
Poliomyelitis, anterior, acute, 797
    in adults, 802
    in children, 798
      pathology, 798
      symptoms, 799
      treatment, 801

Poliomyelitis, subacute and chronic, 803
Polyneuritis, acute febrile, 851
Portal vein, obstruction of, chronic, 603
  thrombosis of, 604
Pox. See *Syphilis.*
Pseudo-angina, 269, 270
Pseudo-leukæmia, 727
  anæmia in, 729
  diagnosis, 730
  etiology and pathology, 727
  glandular swellings in, 729
  symptoms, 728
  treatment, 730
Pseudo-rabies, 168
Pseudo-rheumatism, 675
Pulmonary affections complicating rheumatism, 671
  regurgitation, 242
  stenosis, 243
Purpura, fulminating, 706
  hæmorrhagica, 706
    acute, 706
    subacute, 707
  rheumatica, 705
  symptomatic, 705
Purpuric diseases, 704
    treatment, 709
Pustule, malignant. See *Anthrax.*
Pyæmia, 121
  arterial, 122
  definition, 121
  diagnosis, 123
  etiology, 122
  idiopathic, 122
  prognosis, 123
  symptoms, 123
  treatment, 124
Pyelitis, 656
Pyelonephritis, tubercular, 648
Pyo-pneumothorax, 420
  subphrenic. See *Perihepatitis, acute.*
Pyrosis, 447
Pyuria, 621

Quinine in treatment of epidemic influenza, 92
    of malarial fever, 161

Rabies. See *Hydrophobia.*
  pseudo-, 168.
Rachitis, 701
Râles, pleural and bronchial, differential diagnosis, 402
Raynaud's disease, 899
Rectum, cancer of, 522
    physical examination, 523
    prognosis and treatment, 524
Regurgitation, aortic, 234
  pulmonary, 242
  tricuspid, 241
Relapsing fever, 51
    complications, 54
    convalescence from, 54
    definition and synonyms, 51
    etiology, 52
    exciting cause, 52
    pathology, 52
    prognosis, 54
    symptoms, 53
    treatment, 54
Ren mobilis. See *Kidney, movable.*
Respiratory system, diseases of, 292
Rheumatic fever, 667
Rheumatism, articular, acute, 667
      complications, 670
      etiology, 667
      inflammation of serous membranes in, 669
      symptoms, 668
      theories as to causation of, 667
      treatment, 673
    chronic, 675
      pathology and symptoms, 676
      treatment, 677
  cerebral, 672
  muscular, 677
    treatment, 678
  pseudo-, 675
  secondary, 675
Rheumatisme fibreux, 676
Rickets, 701
  etiology and pathology, 701
  symptoms, 702
  treatment, 704
Roseola. See *Rubella.*
Rötheln. See *Rubella.*
Rubella, 81
  eruption of, 82
  etiology and synonyms, 81
  prognosis, 82
  symptoms, 82
  treatment, 82
Rubeola. See *Measles.*
Rumination, 480

- Sapræmia, 125
- Scarlatina. See *Scarlet fever.*
- Scarlet fever, 66
  - and diphtheria, relation between, 67
  - angina cases, 72
  - complicating inflammations, 68
  - complications, 72
    - kidneys, lesions of, 72
    - muscles, inflammation of, 74
    - serous membranes, inflammation of, 74
  - definition and synonyms, 66
  - desquamation, 70
  - eruption of, 69
  - etiology, 67
  - hemorrhagic cases, 71
  - malignant cases, 71
  - mild and rudimentary cases, 70
  - pathology, 68
  - prognosis, 74
  - severe and prolonged cases, 71
  - susceptibility to, 67
  - symptoms, 68
  - treatment, 74
  - variations from regular course, 70
- Schönlein's disease, 705
- Sciatica, 858
- Sclerema, 904
- Scleroderma, 904
- Sclerosis, amyotrophic lateral, 807
  - cerebro-spinal, 781
  - lateral, 810
    - primary, 810
    - secondary, 811
  - posterior spinal. See *Locomotor ataxia.*
- Scorbutus, 698
- Scrofula, 733
- Scurvy, 698
  - in children, 699
- Sepsis. See *Septicæmia.*
  - acute peritoneal, 537
  - peritoneal, treatment, 543
- Septicæmia, clinical forms, 124
  - definition and synonym, 124
  - progressive, 126
- Seventh nerve, diseases of, 835
- Sinuses, venous, thrombosis of, 760
- Sixth nerve, diseases of, 834
- Small-pox, 55
  - complications and sequelæ, 61
  - confluent form, 59
- Small-pox, contagiousness of, 55
  - definition and synonym, 55
  - discrete form, 56
    - stage of eruption, 57
    - stage of invasion, 56
  - etiology, 55
  - hemorrhagic form, 60
  - malignant form, 60
  - pathology, 56
  - prognosis, 62
  - susceptibility to, 55
  - symptoms, 56
  - treatment, 62
  - varioloid, 61
- Spasm, habit- , 879
- Spinal accessory nerve, diseases of, 844
  - cord, blood-vessels of, anæmia of, 794
      - diseases of, 794
      - hyperæmia of, 794
    - diseases of, 788
    - embolism and thrombosis of, 796
    - hemorrhage into, 795
    - symptoms resulting from acute destruction of anterior cornua of, 798
    - tumors of, 830
  - membranes, hemorrhage into, 793
- Spirillum fever. See *Relapsing fever.*
- Splenic fever. See *Anthrax.*
- St. Vitus's dance. See *Chorea.*
- Steam-tent in treatment of diphtheria, 114
- Stenocardia. See *Angina pectoris.*
- Stenosis, aortic, 239
  - mitral, 230
  - pulmonary, 243
  - tricuspid, 242
- Stomach, atrophy of. See *Gastritis, chronic catarrhal.*
  - cancer of, 464
    - etiology, 464
    - pathology, 464
    - peculiarities in the clinical course, 470
    - physical examination in, 469
    - prognosis, 471
    - symptoms, 466
      - constitutional, 469
      - gastric, 466
      - of secondary growths, 469
  - dilatation of, 452

Stomach, dilatation of, pathology, 452
    physical signs, 454
    prognosis, 455
    symptoms, 453
    treatment, 465
  diseases of, 440
  hemorrhage from, 472
    diagnosis, 474
    etiology, 472
    symptoms, 473
    treatment, 475
  neuroses of, 475
    dyspepsia, nervous, 475
    gastralgia, gastrodynia, 477
    hypersecretion and hyperacidity, 476
    peristaltic unrest, 480
    rumination, 480
    vomiting, nervous, 479
  tumors of, non-cancerous, 471
  ulcer of, 456
    etiology, 456
    pathogenesis, 459
    pathology, 457
    sequelæ and complications, 458
    symptoms, 460
    treatment, 462
    varieties in clinical course, 461
Strongylus duodenalis, 912
Stupor, syphilitic, 787
Sun-stroke, 895
Syncope, local, 899
Syphilides, late, 141
Syphilis, 137
  acquired, 138
    periods of, 138
    prognosis, 142
    symptoms of primary stage, 138
      of secondary stage, 139
      of tertiary stage, 141
    treatment, curative, 143
      prophylactic, 142
  hereditary, 145
    etiology, 145
    symptoms, 146
    treatment, 147
  tertiary, amyloid degeneration following, 142
Syringo-myelia, 828

TABES DORSALIS. See *Locomotor ataxia.*
  mesenterica, 735
Tachycardia, 263
Tænia mediocanellata, 916
  saginata, 916
  solium, 915
Tæniæ, 915
Tape-worms, 915
Tetanilla, 880
Tetanus, 169
  bacillus of, 169
  definition and synonyms, 169
  diagnosis, 171
  duration, 171
  etiology, 169
  hydrophobic, 171
  pathology, 170
  prognosis, 172
  symptoms, 170
  treatment, 172
Tetany, 880
Thermic fever, 896
Third nerve, diseases of, 832
Thomsen's disease, 909
Thread-worms, 911
Thymus gland, diseases of, 431
Thyroid-feeding in treatment of myxœdema, 903
Tic convulsif, 879
  douloureux, 834, 857
  simple, 879
Torticollis, 845
  spasmodic, 845
Trematodes, 910
Tremor cordis, 262
Trichina spiralis, 913
Trichinosis, 913
Trichocephalus dispar, 912
Tricuspid regurgitation, 241
  stenosis, 242
Trismus, 834. See also *Tetanus.*
Tuberculosis, miliary, acute, 148
      definition, 148
      diagnosis, 152
      etiology, 148
      pathology, 148
      prognosis, 152
      symptoms, general, 149
        local, 150
      treatment, 152
      types of the disease, 151
  pulmonary, acute, 373
      diagnosis, 376
      pathology, 374
      physical signs, 375

Tuberculosis, pulmonary, acute, prognosis, 377
    symptoms, 374
    treatment, 395
  chronic, 377
    course, 379
    physical signs, 379
    prognosis, 381
    symptoms, 378, 381
    treatment, 395
 treatment, 395
  creosote in, 398
  Koch's lymph in, 398
  prophylactic, 395
  to arrest the disease, 396
  to relieve symptoms, 399
Tumors of the stomach, non-cancerous, 471
Typhoid fever, 17
  catarrhal or croupous enteritis in, 22
  changes in the intestinal lymphoid tissue in, 21
    in the mesenteric glands in, 22
  complicating lesions, 22
  complications, 35
  convalescence from, 38
    treatment during, 45
  course, 32
  definition and synonyms, 17
  diet in, 40
  eruption of, 31
  essential lesions, 21
  etiology, 17
  exciting cause, 18
  heart-muscle in, 23
  in children, 34
  insidious cases of, 34
  in the aged, 35
  kidneys in, 23
  larynx in, 23
  liver in, 23
  lungs in, 23
  methods of infection, 19
  parotitis in, 23
  pathology, 20
  peritonitis in, 22
  personal susceptibility to, 18
  pharynx in, 23
  prognosis, 38
  pulse in, 26
  relapses and recrudescences, 37
  spleen in, 22, 30
Typhoid fever, symptoms, 23
    fever, 24
    gastro-intestinal, 26
    nervous, 29
    pulse, 26
  treatment, 39
    Brand's method, 42
      contraindications to employment of, 42
    during convalescence, 45
    general management, 40
    internal antipyretics in, 43
    medical, 41
    modified baths, 43
    of recrudescences, 46
    prophylactic, 39
    stimulants in, 44
    wet pack in, 43
  urine in, 31
Typhus fever, 46
  complications, 50
  convalescence from, 50
  crisis in, 50
  definition and synonyms, 46
  eruption in, 48
  etiology, 46
  pathology, 47
  prognosis, 50
  symptoms, 47
    cerebral, 49
  temperature in, 47
  treatment, 51

ULCER, peptic. See *Stomach, ulcer of.*
Unrest, peristaltic, 479
Uric acid, daily excretion of, 622
Uricæmia, 622
Urinary secretion, anomalies of, 614
Urine, acetone in, 625
  albumin in, Heller's test for, 616
  blood in, Heller's test for, 618
  changes in, in chronic Bright's disease, 637, 641
  chyle in, 624
  diminished, in acute degeneration of the kidneys, 628
    in congestion of the kidneys, 626, 627
    in movable kidney, 613
  fat in, 625
  hæmoglobin in, 619
  in acute exudative nephritis, 630, 631

Urine in acute yellow atrophy of the liver, 562
  in diabetes mellitus, 693
  indigo in, 624
  in gout, 683
  in jaundice, 591
  in tubercular pyelonephritis, 648
  in waxy degeneration of the kidneys, 647
  oxalate of lime in, 624
  peptone in, 621
  phosphates in, 622
  pus in, 621
  quantity of, in chronic degeneration of the kidneys, 629
  uric acid in, 622
Urticaria, giant, 900

VACCINATION. See *Vaccinia.*
  complications of, 65
Vaccinia, 64
Valvular disease. See *Endocarditis, chronic.*
Varicella, 65
  definition and synonym, 65
  eruption of, 66
  etiology, 65
  prognosis, 66
  symptoms, 65
  treatment, 66
Variola. See *Small-pox.*
Varioloid, 61
Vaso-motor and trophic disorders, 899
Vertigo, auditory, 839
  labyrinthine, 839
Vomiting in acute peritonitis, 540
Vomiting in cancer of the stomach, 467
  in cholelithiasis, 597
  in intestinal obstruction, 528
  nervous, 479

WATER-BRASH, 447
Weil's disease, 181
Werlhoff's disease, 706
Westphal's symptom, 815
Whip-worm, 912
Whooping-cough, 85
  complications and sequelæ, 87
  definition and synonym, 85
  etiology, 85
  lesion, 85
  symptoms, 85
  treatment, 87
    local, 88
Woolsorter's disease. See *Anthrax.*
Wrist-drop, 851
Writer's cramp, 884
Wry-neck, 845

YELLOW FEVER, 133
  complications and sequelæ, 136
  contagiousness of, 134
  definition, 133
  diagnosis, 136
  etiology, 133
  immunity from, 134
  incubation, 134
  pathology, 134
  prognosis, 136
  symptoms, 134
  treatment, general, 137
    prophylactic, 136

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